Hot Topics in Acute Care Surgery and Trauma

Federico Coccolini
Rao Ivatury
Michael Sugrue
Luca Ansaloni *Editors*

Open Abdomen

A Comprehensive Practical Manual

Springer

Hot Topics in Acute Care Surgery and Trauma

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Open Abdomen

A Comprehensive Practical Manual

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Foreword to the Series

Since 2011, the founding members of the World Society of Emergency Surgery (WSES) Acute Care and Trauma Surgeons group, in collaboration with the American Association for the Surgery of Trauma (AAST), endorse the "Hot Topics in Acute Care Surgery and Trauma," realizing the need to provide more educational tools for young in-training surgeons and for general physicians and other surgical specialists. These new forthcoming titles have been selected and prepared with this philosophy in mind. The books will cover the basics of pathophysiology and clinical management, framed with the reference that recent advances in the science of resuscitation, surgery, and critical care medicine have the potential to profoundly alter the epidemiology and subsequent outcomes of severe surgical illnesses and trauma. In particular, **open abdomen** requires detailed understanding as to the potential benefits and complications associated with this surgical approach. There exists, implicit in use of the open abdomen approach, a decision to adopt the damage control paradigm in the management of the sickest patients. No other management approach has been so profoundly linked to changes in resuscitation, with recent advances prompting a reassessment and critique of all previously accepted principles.

Cesena, Italy — Federico Coccolini
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Foreword

It is an honor and great pleasure to write a foreword to the book *Open Abdomen: A Comprehensive Practical Manual* edited by surgeons and professors Federico Coccolini, Rao Ivatury, Michael Sugrue, and Luca Ansaloni. Textbooks focusing on abdominal wall complications and hernia techniques do exist, but it is still a challenge to treat an open abdomen patient who needs an abdominal wall reconstruction. In the era of damage control surgery, complex abdominal wall defects are more common. Most patients have consequences of complex trauma, cancer, and necrotizing infections, resulting in abdominal catastrophes without an abdominal wall.

New technologies are coming—vacuum-assisted systems have been used in different countries; meshes and biological prostheses developed with biomedical research have changed the options for surgical repair. The approach to treat patients with open abdomen or complex abdominal wall defect is multidisciplinary, and team experience is important to have good results, with less complications.

This book presents, in its 22 chapters, the multiple aspects of the challenging open abdomen and abdominal wall reconstruction. It covers the topic from its beginning with historical hints; the anatomy; pathophysiology; indications; management in infants, children, and adults; nutritional aspects, complications; definitive closure; the use of biological prostheses; and quality of life. The editors were very competent in bringing together a select group of surgeons and teachers who present the subjects in an objective and practical way, facilitating the learning of the readers.

There is an old aphorism in medicine that says “the surgeon should not be the first to adopt a new technique and not be the last to abandon an old technique.” This manual exhaustively describes when “to leave open and when to close” an abdomen. These two key issues are analyzed based on the latest evidence in literature, with the support of a strong tool: the WSES Guidelines recently published. This book brings content in the right dose, helping the next generation of surgeons to properly treat their patients, and it is a gift to the readers and even more to their patients who will benefit from a more appropriate treatment.

Campinas, SP, Brazil G. P. Fraga
Parma, Italy F. Catena

Preface

The treatment of patients with intra-abdominal catastrophes with an open abdomen involves several potential complex management strategies. The open abdomen is universally recognized as an option to improve outcomes in selected patients. Firstly described in damage control procedures in trauma the open abdomen is now increasingly used in nontrauma patients. The challenge and key in decision making is balancing whether an open abdomen is going to benefit patients yet not create counter-product morbidity. This book explores indications for an open abdomen and offers practical tricks and techniques to minimize the side effects.

The key to the open abdomen is to allow the patient's physiology to return to normal. Comprehending the pathophysiological mechanisms behind its application is essential and explained in the book. The manual presents different points of view from the clinicians involved in the management of the open abdomen patients. It explores care from the very beginning of the treatment through to rehabilitation. This will help the reader integrate the best information into their practice, from recognized experts in their respective fields.

We would like to thank all the authors for sharing their precious experience and hope it will enhance your patient's outcome.

Cesena, FC, Italy — Federico Coccolini
Richmond, VA, USA — Rao Ivatury
Letterkenny, Ireland — Michael Sugrue
Cesena, Italy — Luca Ansaloni

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Open Abdomen: Historical Notes

1

Rao R. Ivatury

The story of "open abdomen" management (OAM) is reminiscent of many other advances in medicine: described, forgotten, reinvented, ridiculed, and finally accepted. The science of OAM continues to unfold and presents us with a fascinating glimpse into the mysteries of pressure–perfusion phenomena, cytokine response to injury and sepsis, the splanchnic bed, and its role as the motor for multiorgan failure, just to name a few. It is a rational surgical approach, based on solid physiologic principles. Many of the details of OAM management will be discussed in detail in other parts of this work. This chapter will present the evolutionary history of OAM from its origin to the current state.

OAM may very well be an apt example of the oft-quoted adage: "There is very little new under the sun." All the way back in 1940, Sir Heneage Ogilvie already devised the use of a "double sheet of light canvas or stout cotton cut rather smaller than the defect in the muscles, and sutured into place with interrupted catgut sutures" for temporary closure of abdominal war wounds when there was too much tension to close primarily [1]. He also described the use of Vaseline-impregnated gauze rolls over exposed viscera and closure of wound over them with strips of Elastoplast® or stitches. In a subsequent report, Ogilvie advocated the same technique to leave the abdomen open and close it secondarily after 1–4 days [2]. He described it as akin to draining an abscess by open drainage.

This great innovation, though well described, was soon forgotten for another 40 years. Tables 1.1 and 1.2 are a chronologic summary of landmark articles from 1940 to 2000. Steinberg [4] resurrected the concept in 1979. He described treating the purulent abdomen as an abscess, incising it and leaving it open. In 14 patients with acute generalized peritonitis, the abdomen was left open after the first laparotomy by gauze packs on the viscera. Abdominal wires were placed to be tied to close the abdomen after 48–72 h. Only one of the 14 died. Another developed an

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Table 1.1 Literature reports of the first 50 years of open abdomen (1940–1990) for abdominal sepsis

Author, year	Indication for OAM	Results	Comments
Ogilvie, 1940	Temporary closure of abdominal war wounds with too much tension to close primarily		"Double sheet of light canvas or stout cotton cut rather smaller than the defect in the muscles and sutured into place with interrupted catgut sutures" "Vaseline-impregnated gauze rolls over exposed viscera and closure of wound over them with strips of Elastoplast® or stitches"
Ogilvie, 1945	Leave the abdomen open and close it secondarily after 1–4 days		
Steinberg, 1979	Fourteen patients with acute generalized peritonitis, the abdomen was left open after the first laparotomy by gauze packs on the viscera. Abdominal wires were placed to be tied to close the abdomen after 48–72 h	Mortality 7%	"Treat the infected abdomen just as an abscess"
Duff and Moffat, 1981	Abdominal sepsis, necrotizing wound infection	39% mortality	Abdominal closure by STSG/skin sutures
Mughal, 1986	Fourteen patients: GI perforations/anastomotic dehiscence (n-11), recurrent pancreatic abscess ($n = 3$)	28% mortality	OAM termed "laparostomy"
Hedderich, 1986	Ten patients with abdominal sepsis and fecal peritonitis (3), radiation enteritis with fistula (1), diverticular abscess (1), diffuse post-op sepsis (2), necrotizing pancreatitis (3)	20% mortality	Marlex mesh with zipper
Schein, 1988	Nine with diffuse peritonitis	32% mortality	Average seven reoperations per patient!
Mastboom, 1989	Fourteen with 53 small bowel fistulas from OAM	64% mortality	A detailed description of this dreadful complication after OAM
Garcia-Sabrido, 1989	Forty-nine with necrotic pancreatitis Fifteen with severe intra-abdominal sepsis	Intra-abdominal sepsis: (in 26.5%) mortality (expected 45%), pancreatic necrosis (6%, single abscess, 9, 22% infected pancreatic necrosis (expected 47%)	Quantification of severity of illness by APACHE II

Ivatury, 1989	Thirty patients with abdominal sepsis (11 patients after trauma [group 1], 5 pancreatic abscess [group 2], 14 acute GI pathology [group 3])	Mortality 27% in group 1, 40% in group 2, and 64% in group 3	Survival: age < 50 years and the absence of multiple organ failure Quantification of severity of illness by APACHE II Absorbable mesh for TAC
Ivatury, 1990	13 with post-trauma abdominal sepsis, 11 with MOF	Mortality 23%	APACHE II scoring, absorbable mesh for TAC
Whitmann, 1990	One hundred and seventeen treated by "Etappen lavage"	APACHE II scoring Actual mortality 25% (expected mortality 47%)	TAC retention sutures ($n = 45$), a simple zipper ($n = 26$), a slide fastener ($n = 29$), and Velcro analogue ($n = 17$)
Christou, 1993	Two hundred and thirty-nine patients with surgical abdominal infection in a prospective, open, consecutive, nonrandomized trial	"Closed abdomen technique": 31% mortality versus 44% open abdomen	Surgical Infection Society study

Table 1.2 Literature reports, 1990–2000, on damage control surgery (DCS) for trauma

Author, year	Indication for OAM	Comments
Burch, 1992	Abbreviated laparotomy	"Multiple towel clip" closure of abdominal incision, not truly an OAM technique
Morris, 1993	Staged celiotomy for trauma	"Rapid closure of the abdomen" in phase 1. Decompressive laparotomy for ACS. Not truly an OAM technique. ACS is diagnosed when fully developed
Rotondo, 1993	"Damage control" (DC): first use and description of term	"Rapid closure of the abdomen." Not truly an OAM technique, no mention of IAP measurement
Meldrum, 1997	Twenty-one (14%) of 145 patients (ISS > 15) requiring laparotomy developed ACS; 24% were planned decompression. The remaining were prompted by deteriorating organ function from ACS	Not truly an OAM technique. ACS is diagnosed only when fully developed Proposed ACS grading system for selective management of the syndrome
Ivatury, 1997	State-of-the-art review article	Emphasis on IAP measurement, IAH as a precursor of ACS, surgical intervention at the stage of IAH and not wait until full-blown ACS Among the first to propose non-closure of fascia, OAM with fascial prosthesis as prophylaxis for IAH in high-risk patients
Mayberry, 1997	Group 1 consisted of 47 patients who received mesh at initial celiotomy, and group 2, 26 patients who received mesh at a subsequent celiotomy. Groups were statistically similar in demographics, injury severity, and mortality. Group 2 had a significantly higher incidence of postoperative abdominal compartment syndrome (35 versus 0%), necrotizing fasciitis (39 versus 0%), intra-abdominal abscess/peritonitis (35 versus 4%), and enterocutaneous fistula (23 versus 11%) compared with group 1 ($p < 0.001$)	Among the first to emphasize prophylaxis for ACS with absorbable mesh in the fascia

Table 1.2 (continued)

Author, year	Indication for OAM	Comments
Ivatury, 1998	In patients with severe penetrating abdominal trauma having DCS, injury severity was comparable between patients who had mesh closure as prophylaxis for IAH ($n = 45$) and those who had fascial suture ($n = 25$). IAH was seen in 10 (22.2%) in the mesh group versus 13 (52%) in the fascial suture group ($p = 0.012$) for an overall incidence of 32.9%. Forty-two patients had pHi monitoring, and 11 of them had IAH. Of the 11 patients, 8 patients (72.7%) had acidotic pHi (7.10 ± 0.2) with IAH without exhibiting the classic signs of ACS. The pHi improved after abdominal decompression in six and none developed ACS. Only two patients with IAH and low pHi went on to develop ACS, despite abdominal decompression. Multiorgan dysfunction syndrome points and death were less in patients without IAH than those with IAH and in patients who had mesh closure	IAH may cause gut mucosal acidosis at lower bladder pressures, long before the onset of clinical ACS. Uncorrected, it may lead to splanchnic hypoperfusion, ACS, distant organ failure, and death Prophylactic mesh closure of the abdomen may facilitate the prevention and bedside treatment of IAH and reduce these complications "Leave the abdomen open!"
Sugrue, 1998	Prospective study of outcomes in 49 consecutive patients undergoing TAC for decompression in 22, inability to close the abdomen in 10 and to facilitate re-exploration for sepsis in 8 and multifactorial in 9 patients	After TAC, there was a significant reduction in IAP, improvement in lung dynamic compliance and brisk diuresis. There was no significant improvement in renal function or patient oxygenation
Ertel, 2000 [3]	European study. 17 of 311 patients (5.5%) having DCS developed ACS because of persistent intra-abdominal/retroperitoneal bleeding ($n = 12$; 70.6%) or visceral edema ($n = 5$; 29.4%)	Primary fascial closure, no OAM till the development of ACS

intra-abdominal abscess. Duff and Moffart [5] described OAM for severe, uncontrolled abdominal sepsis with or without necrotizing abdominal wound infection. They observed a mortality rate of 39% and noted the benefits of the OAM. Maetani and Tobe [6] also reported in support of "open peritoneal drainage" in 13 patients with advanced peritonitis. Mughal et al. [7] called the OAM "laparostomy" and reported on 18 patients with severe sepsis or acute necrotizing pancreatitis. The overall mortality was 28%. They concluded that laparostomy is a valuable technique in the management of severe, intractable intra-abdominal sepsis. Schein et al. [8–10] published a series of articles on OAM from 1986 to 1988 and correctly predicted all the problems with this approach, to include spontaneous fistulae, exogenous bacterial contamination, evisceration, massive fluid losses, need for ICU care, and subsequent closure of the abdomen. Garcia-Sabrido et al. [11] used OAM (zipper alone or a zipper-mesh combination) with the provision for daily laparotomy in the intensive care unit with the patient under epidural anesthesia. For the first time,

a quantification of severity of illness by APACHE II scores and observed versus expected mortality were introduced: expected mortality of 45% versus the 26.5% observed mortality. Other support for OAM [12, 13] continued to be published, even though some detractors would emphasize all the potential morbidity. Mastboom et al. [14] published the first large experience with small bowel perforation complicating the open treatment of generalized peritonitis and suggested that the open abdomen itself is a risk factor for this complication.

In 1987, Ivatury and colleagues from the Bronx presented to the New York Surgical Society their experience with OAM, reporting results according to primary septic pathology and quantifying severity of illness [15]. Thirty patients with uncontrolled abdominal sepsis, 1982–1987 (11 patients after trauma [group 1], 5 patients with pancreatic abscess [group 2], and 14 patients with acute GI pathology [group 3]), and worsening organ functions had OAM management. Sixteen (53%) of the 30 patients survived, 73% in group 1, 60% in group 2, and 36% in group 3. Survival correlated well with age less than 50 and the absence of multiple organ failure. The authors emphasized that the technique was easy to perform, avoiding many of the pitfalls previously reported. Temporary abdominal closure (TAC) was achieved with an absorbable mesh for the first time. They pointed out that the absorbable polyglycolic acid (Dexon®) was found superior to the nonabsorbable polypropylene mesh.

In 1989, this group presented their second series to the Eastern Association for the Surgery of Trauma and published it in 1990 [16]. Thirteen patients with abdominal trauma who developed abdominal sepsis resistant to conventional methods of re-exploration and drainage were managed by OAM. Eleven of these patients had multiorgan failure. Ten of the 13 patients (76.9%) survived, a significantly improved survival as compared with that predicted by APACHE (50%). This group continued to develop their OAM technique, changing TAC prosthesis from Dexon® to a Vicryl® mesh. They documented a reduced need for assisted ventilation and a decreased ICU stay. Tolerance of enteral feeding, ambulation, reduction of complications, and increasing survival were all noted with increasing experience with the OAM technique. In the early 1990s, however, definitive fascial closure was not as much a pursued outcome as having survival from the serious illness that required OAM. The ultimate result of the OAM was an abdominal wall hernia, to be repaired in willing patients much later in another admission.

Other novel techniques of avoiding OAM were introduced by others in the early 1990s: “sandwich technique” [9], “Etappen lavage,” or planned relaparotomy [17]. Some kept the abdomen closed in between procedures; others used various closure techniques such as retention sutures, slide fasteners, zippers, and Velcro adhesive sheets or towel clips.

Some of the enthusiasm for OAM was dampened by lack of controlled trials. The sight of exposed bowel loops under the TAC was abhorrent to many surgeons, and they were fearful of perceived potential for complex postoperative complications. This discouraged them from trying the technique. Groups who were used to it, however, were greatly enthusiastic. In 1993, the Surgical Infection Society carried out a prospective, open, consecutive, nonrandomized trial to examine management

techniques and outcome in severe peritonitis [18]. A total of 239 patients with surgical infection in the abdomen were studied. There was no significant difference in mortality between patients treated with a "closed abdomen technique" (31% mortality) and those treated with variations of the "open abdomen" technique (44% mortality). This was another setback for the OAM technique. Another randomized trial done in 2007 also failed to find a survival advantage with OAM, essentially making it an unpopular method of treating abdominal sepsis [19]. The difficulty of the decision and timing to perform a relaparotomy in the on-demand strategy for intra-abdominal sepsis was largely ignored by the detractors of OAM. Factors indicative of progressive or persistent organ failure during early postoperative follow-up were shown to be the best indicators for ongoing infection and were associated with positive findings at relaparotomy [20]. Planned relaparotomy did not, therefore, lose its indication for selected patients. Amazingly, the fact that OAM achieved the same goal without sacrificing the fascia from the trauma of repeated opening and closing was not accepted by these detractors of OAM.

The current status of OAM in abdominal sepsis will be revisited later in the chapter.

1.1 The Era of OAM, "Damage Control" Surgery (DCS), Intra-abdominal Hypertension (IAH), and Abdominal Compartment Syndrome (ACS)

The 1990s saw an escalation of major blunt and penetrating trauma in US trauma centers across the country began to deal with increasing number of patients with increasing severity of anatomic and physiologic injury. A majority of these patients were being seen at the end of their physiologic reserve, a situation called "physiologic exhaustion." Severe hemorrhagic shock, acidosis, need for massive resuscitation fluids, ensuing hypothermia, and coagulopathy leading to more bleeding from nonsurgical sources, essentially a vicious circle of ischemia–reperfusion injury and its consequences, were becoming a nightly challenge. Something needed to be strategized to interrupt this vicious cycle. It is in this environment that trauma centers began to apply the principles of abbreviated initial laparotomy to control major hemorrhage and enteric contamination, temporary abdominal closure, resuscitation in the ICU to reasonable physiologic restoration (improving base deficit, serum lactate, coagulation parameters), and subsequent return to the operating room to complete organ repairs, bowel anastomoses, abdominal closure, etc. Rotondo et al., in 1993, gave tremendous impetus for the promulgation of these concepts when they christened the process "damage control surgery" [21]. This truly heralded a new era in the management of the most severely injured and ill patients.

Inevitably, other aspects of this phenomenon soon followed: OAM, temporary abdominal closure (TAC), intra-abdominal hypertension (IAH), abdominal compartment syndrome (ACS), and subsequent escalation of damage control surgery (DCS) philosophy to increasingly complex abdominal, thoracic, peripheral, vascular, and orthopedic injuries. Roberts et al. [22] recently published a fascinating

historical account of DCS that should be studied by all that are interested in trauma. They showcased how previously abandoned surgical techniques (e.g., peri-hepatic packing, leaving the abdomen open) were resurrected in response to our enlightened concepts of trauma patient physiology.

DCS with emphasis on abbreviating laparotomy, leaving intra-abdominal packing to control nonsurgical bleeding and evacuation of the patient in physiologic vortex out of the operating room, is the recipe that demanded non-closure of the abdomen and TAC by other means so that aggressive resuscitation can take place in the ICU. Other resuscitation practices that were prevalent at this time also created a need for the OAM. Specifically, the practice of supranormal oxygen delivery as an endpoint of adequacy of resuscitation, even though debunked by two prospective trials [23, 24], meant excessive crystalloid and colloid infusion. This lead to excessive peritoneal fluid, tissue edema, and problems related to increased intra-abdominal pressure (IAP) or intra-abdominal hypertension (IAH) leading to the full-blown abdominal compartment syndrome (ACS). A whole new science of pressure–perfusion phenomena in the OAM was thus born.

In the early 1990s, several distinguished groups were already preaching about the ill effects of IAH that were learned in the animal laboratories in the early 1970s [25]. The profound implications of IAH on diverse organ systems were increasingly discussed, and the prominent publications [26–32] are chronologically summarized in Table 1.2. Collectively termed as abdominal compartment syndrome (ACS), these constellation of physiologic aberrations were gaining notoriety as complications that should be recognized and avoided. A state-of-the-art review was published by Ivatury et al. in 1997 [29], emphasizing OAM as prophylaxis against ACS. The following two studies provided confirmatory evidence for these concepts.

Mayberry and associates, in 1997 [30], reported on 73 consecutive trauma patients requiring celiotomy and receiving absorbable mesh prosthesis closure for excessive fascial tension, ACS, necrotic fascia, or a planned reoperation. Group 1 consisted of 47 patients who received mesh at initial celiotomy, and group 2, 26 patients who received mesh at a subsequent celiotomy. These two groups were statistically similar in demographics, injury severity, and mortality. However, group 2, compared with group 1, had a significantly higher incidence of postoperative abdominal compartment syndrome (35 versus 0%), necrotizing fasciitis (39 versus 0%), intra-abdominal abscess/peritonitis (35 versus 4%), and enterocutaneous fistula (23 versus 11%) ($p < 0.001$). They concluded that the use of absorbable mesh prosthesis closure in severely injured patients undergoing celiotomy was effective in treating and preventing ACS.

Ivatury and associates [31] had been studying patients with catastrophic penetrating trauma undergoing damage control procedures from 1992 to 1996. They presented their observations at the annual scientific assembly of the American Association for the Surgery of Trauma (AAST) in 1997. Seventy patients in the SICU at their Level I trauma center had intra-abdominal pressure estimated by bladder pressure. Gastric mucosal pH (pHi) was measured by gastric tonometry every 4–6 h. IAH (intra-abdominal pressure > 25 cm of H2 O) was treated by bedside or

operating room laparotomy. Injury severity was comparable between patients who had OAM with mesh closure as prophylaxis for IAH (n = 45) and those who had fascial suture (n = 25). IAH was seen in 10 (22.2%) in the mesh group versus 13 (52%) in the fascial suture group (p = 0.012) for an overall incidence of 32.9%. Forty-two patients had pHi monitoring, and 11 of them had IAH. Of the 11 patients, 8 patients (72.7%) had acidotic pHi (7.10 ± 0.2) with IAH without exhibiting the classic signs of ACS. The pHi improved after abdominal decompression in six patients and none developed ACS. Only two patients, both with extreme bowel thickening and edema, had IAH and low pHi, went on to develop ACS, and died despite abdominal decompression. Multiorgan dysfunction syndrome points and death were less in patients without IAH than those with IAH and in patients who had mesh closure. The authors concluded: "IAH was frequent after major abdominal trauma. It may cause gut mucosal acidosis at lower bladder pressures, long before the onset of clinical ACS. Uncorrected, it may lead to splanchnic hypoperfusion, ACS, distant organ failure, and death. Prophylactic mesh closure of the abdomen may facilitate the prevention and bedside treatment of IAH and reduce these complications." Two important concepts were thus advanced:(1) IAH is the prodrome of ACS and monitoring of IAP in these high-risk patients, and prompt intervention at the stage of IAH may prevent the full syndrome of ACS with its attendant morbidity and mortality, and (2) in patients with extensive abdominal injuries undergoing DCS, "non-closure of the fascia," leaving the abdomen open with a fascial prosthesis, may prevent ACS, reduce organ failures, and lead to better survival. Soon, this was accepted as an integral part of DCS principles [33]. Even though temporary abdominal closure (TAC) varied in its type and form (as discussed below), OAM became an integral part of initial laparotomy of DCS.

Our knowledge of IAH and ACS continued to be spurred by the shared experiences of trauma centers dealing with the nightly horrors of "America's uncivil war" as Schwab addressed them in his presidential address [34]. The phenomena were codified by trauma surgeons, who soon popularized the clinical principles of IAP monitoring by bladder pressure and non-closure of fascia after laparotomy ("open abdomen"). The ensuing results were nothing short of dramatic [33]. Further advances were also realized through the efforts of a remarkable group of clinical researchers interested in the subject. After a preliminary meeting in 2001 in Sydney, Sugrue and associates formally established the World Society of the Abdominal Compartment Syndrome (WSACS) in 2004 in Noosa in Australia. This Society, though a small group of motivated clinicians, redefined the current concepts of IAH and ACS through multinational clinical trials; literature review and analysis; multiple publications, including a monograph on the subject [35]; and guideline and consensus development [36–38]. The efforts of anticipation of the complication, measures of prophylaxis, and earlier recognition and intervention all soon bore fruits: fewer organ failures and better survival. In a prospective, observational study, Cheatham and Safcsak [39, 40] studied 478 consecutive patients who were treated with open abdomen for IAH and ACS according to "a continually revised management algorithm" and noted a significantly increased patient survival to hospital discharge from 50 to 72% (p = 0.015) and an increase in same-admission primary

fascial closure from 59 to 81% over the period of the study. This was one of the first clinical series showing that a management focus on IAP can have better outcomes without increasing resource utilization. They also documented that abdominal decompression does not prevent return to gainful employment and should not be considered a permanently disabling condition. Balogh, another executive committee member of WSACS, with his associates [41] prospectively analyzed 81 consecutive severely injured shock/trauma patients (mean ISS 29). No patient developed ACS, even though 61 (75%) had IAH. Multiorgan failure occurred in one patient without IAH (5%) versus four with IAH (7%). The authors commented that monitoring and intervening for a less serious IAH, a practice that resulted in the avoidance of the deadly ACS, was a remarkable triumph of critical care. One should also thank the DCS strategies and OAM for this success.

Other benefits of the understanding of IAH and ACS soon lead to a moderation of the enthusiasm for "hyper" resuscitation in multiple trauma and burns. Dr. Pruitt's call against "fluid creep" [42] was heeded by the turn of the decade. Restricted fluid resuscitation became an important part of the components of "damage control resuscitation," an improved concept of DCS [43] and enhanced survival.

The saga of IAH and ACS, however, once again sadly exemplified the tribulations of medical history: ignored concepts, forgotten lessons, and detraction by "disbelievers." While established trauma centers and academic institutions were eliminating ACS by aggressive application of the concepts narrated here, the paradigm was not received widely. Many examples abound, but some recent ones are given here: the 2013 survey of WSACS with 13 questions to 10,000 members of the WSACS, the European Society of Intensive Care Medicine (ESICM), and the Society of Critical Care Medicine (SCCM) demonstrated that only 28% were aware of the WSACS consensus definitions. Overall knowledge scores were low (43 ± 15%) [44]. Another study [45] surveyed Dutch surgeons with a literature-based and expert consensus survey. Many of these surgeons exhibited a good knowledge of IAH and ACS, but only 27% used this in their daily practice. Another survey [46] found similar ignorance about IAH among Chinese intensive care physicians in tertiary hospitals. A similar lack of application of definitions and guidelines was reported among German pediatric intensivists [47] and Australian critical care nurses [48]. Let us hope that the pioneering work of groups such as the WSAC and the World Society of Emergency Surgery (WSES) will shatter this darkness of ignorance and enlighten all the important groups for the betterment of the patients.

1.2 Temporary Abdominal Closure (TAC) Techniques

Temporary abdominal closure is an integral part of the OAM. It is not surprising that the final result of OAM is a reflection of the design and materials used for TAC. An ideal TAC would minimize nursing problems, control fluid loss, and prevent injury to the viscera. In addition, it should, in some way, facilitate closure of the fascia once the OAM was no longer needed. In most of the existing treatment strategies, the abdomen needed to be closed within a window of 5–7 days for a high chance of fascial closure.

The state of the art in early experience with OAM was TAC with an absorbable mesh, waiting for the abdomen to be covered with healthy granulation tissue. Subsequently it received skin grafting or skin-only closure (Figs. 1.1, 1.2, 1.3, 1.4, 1.5, 1.6, 1.7). In those initial years, two important and highly morbid complications of this approach were frequent: abdominal wall hernia and enteroatmospheric fistula [49–54]. Even though fistula rates were reduced to about 5% by groups with a large experience in OAM, it was still a major problem when it occurred. The ideal TAC device, therefore, not only needed to prevent loss of abdominal domain and fistulization but also to preserve the fascia/abdominal wall integrity to achieve better primary fascial closure rates, while preventing IAH or the development of ACS [50–54].

Many different techniques have been introduced during the past 10 years [49], but there were no controlled trials. Patient groups and pathology were heterogeneous. Comparison of techniques and outcomes was impossible. However, one

Fig. 1.1 TAC with gauze dressings

Fig. 1.2 TAC with towel clips

Fig. 1.3 TAC with Bogota (Borraez) bag

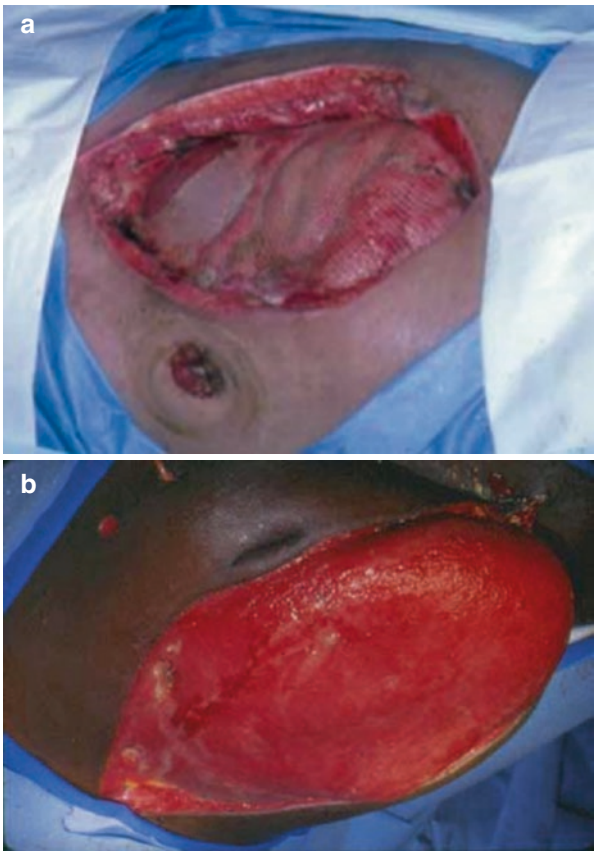

Fig. 1.4 (**a**) TAC with absorbable mesh. (**b**) Granulation tissue covering the entire open abdomen. The mesh was absorbed

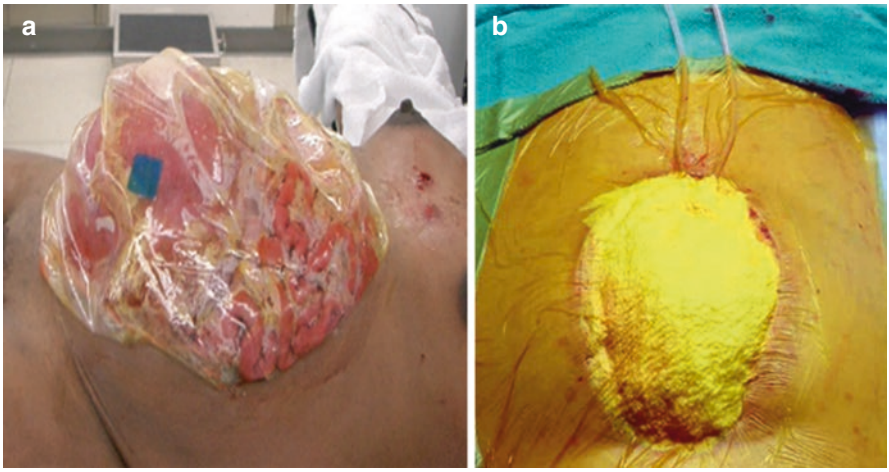

Fig. 1.5 (**a, b**) TAC with vacuum pack

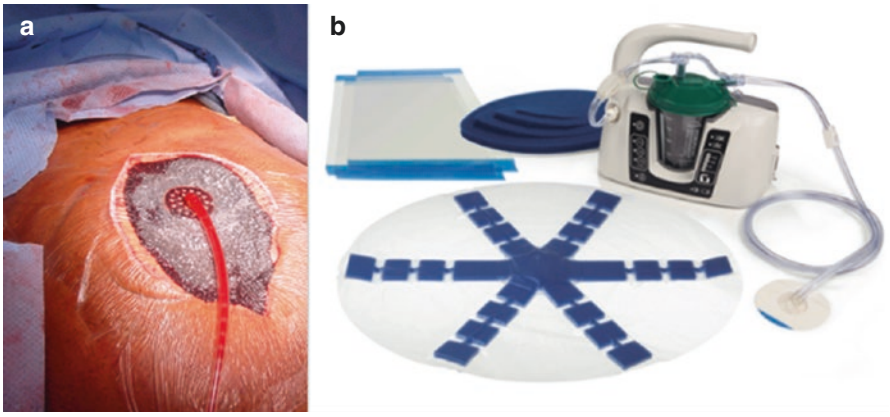

Fig. 1.6 (**a, b**) TAC with commercial VAC and "AbThera"

Fig. 1.7 TAC with dynamic retention suture system

systematic review by Boele van Hensbroek and colleagues [49] suggested that the highest fascial closure rates were seen in the artificial bur (90%), dynamic retention sutures (DRS) (85%), and VAC® (60%) and the lowest mortality rates were seen in the artificial bur (17%), VAC® (18%), and DRS (23%). Here is a brief summary of the TAC techniques that evolved over the past two decades:

1.2.1 The Bogota (Borraez) Bag

Suturing a 3-L urologic irrigation bag to the fascia or skin was first used simultaneously in several institutions in Colombia in 1984 and introduced by Oswaldo Borraez from Bogota. This technique was cheap and easily available and served most of the purposes of TAC. It did not, however, preserve the fascia and did not prevent IAH. In a systematic review, it showed a weighted mortality rate of 41% [49].

1.2.2 Fascial Prosthetic Mesh

Suturing of a mesh was one of the original techniques that was practiced by our group in the late 1980s and early 1990s. Initially a nonabsorbable mesh (polypropylene) was used but soon fell out of favor because of the rigidity, propensity to cavuse bowel fistula when it came into contact with bowel, and also subsequent fragmentation. It did not always prevent hernia formation. Furthermore, it often required a difficult reoperation to excise it from the wound. Absorbable mesh soon became popular, initially polyglycolic acid (Dexon®) and later Vicryl®. These were very supple and handled suturing to the fascia well. They got absorbed and incorporated into the granulation tissue covering the open abdomen. They also were thought to possess antibacterial properties. They did not, however, prevent subsequent ventral hernia. Several notable groups also would use them as a TAC in cases where fascial closure was not possible and a ventral hernia was being accepted [54, 55]. The use of other prosthetic mesh such as GORE-TEX® for TAC has also been reported in some studies [56].

1.2.3 Artificial Bur Device or Wittmann Patch®

This technique was first described in 1990 [17] and improved into the commercially available Wittmann Patch® (NovoMedicus, Germany). The Wittmann Patch consists of hook-and-loop (Velcro®-like) sheets that are pressed together to form a secure closure and peeled apart for abdominal reentry. The overlap is adjusted to accommodate an increase or decrease in swelling. Applying a slight tension helps prevent lateral retraction. As abdominal swelling decreases, the fascial edges are pulled closer together and excess patch material is trimmed. When the two fascial edges are close enough, the remaining patch material is removed, and the abdominal wall is closed by suturing fascia to fascia. In the systematic review [49], it had the highest fascial closure rate (90%).

1.2.4 Dynamic Retention Sutures (DRS)

These are combinations of techniques combined with retention sutures as well as specially designed dynamic retention suture systems (e.g., ABRA®). An RCT comparing a combination of VAC® and retention sutures with VAC® alone in a total of 30 patients with abdominal sepsis achieved significantly higher closure rates in patients treated with the combination (93.3%) [57].

1.2.5 Negative Pressure Therapy Techniques: Vacuum Pack

The principles of these systems focus on the understanding that primary fascial closure within the initial admission is associated with the best outcome, prevention of adhesions between the wall and bowel loops, preserving peritoneal space, evacuating the cytokine-rich peritoneal fluid efficiently and keeping IAP down to minimize organ failures, and preventing contact between the gauze dressings and bowel to avoid development of enteroatmospheric fistulae [58, 59].

Brock in 1995 [58] and Barker in 2007 [59] pioneered the concept of using a vacuum drainage of the free peritoneal fluid by suction catheters. The open abdomen was covered by a fenestrated polyethylene sheet between the abdominal viscera and the anterior parietal peritoneum; a moist, surgical towel over the sheet with two suction drains; and an adhesive drape over the entire wound which is airtight. As soon as the drains were connected to wall suction, the entire apparatus would "collapse," evacuating the peritoneal fluid and blood. We employed a similar technique (figure) as a "poor-man's VAC." In the systematic review mentioned above [49], vacuum pack showed a 52% primary fascial closure rate.

1.2.6 Negative Pressure Therapy Techniques: Vacuum-Assisted Closure and VAC®

This was a first-generation negative pressure therapy system for the OAM introduced to the market in 2003 by KCI USA and made of a fenestrated nonadherent layer with encapsulated foam that is placed on the wound surface; a VAC® Perforated GranuFoam™ Dressing that is placed over the covered wound surface; a plastic drape, which is applied over the abdomen; and the SensaT.R.A.C.™ Tubing that is placed over a hole cut in the drape to have direct contact with the GranuFoam™ dressing and connected to a suction apparatus providing a continuous negative pressure at −125 mmHg. This apparatus purportedly produced a medial tension and removed abdominal fluids, protecting the OAM from external contamination and helping approximate wound margins [49, 60–64].

The importance of a rigid protocol and a standardized approach were illustrated by several reports [60–63] with a fascial closure rate of 88–100%, even as late as 9–21 days after the first laparotomy. Fistula rates were low. Subsequent prospective studies also confirmed the use of VAC® to be safe in septic patients [64, 65].

ABThera™ OAM: This was a second-generation negative pressure therapy system for the OAM and was in introduced by KCI to the market by 2009. It was similar in design to the previous system with the addition of six foam extensions radially situated on the visceral protective layer. They provide for improved fluid removal. A nonadherent fenestrated polyurethane sheet separates the bowel from abdominal wall and helps remove fluid. The ABThera™ Perforated Foam, which delivers negative pressure, provides medial tension to help minimize fascial retraction and loss of domain.

There was great enthusiasm for this improved VAC systems, and they were extensively used and investigated. Two important prospective studies are summarized below.

Cheatham et al. [66] reported on a prospective, multicenter, observational, open-label study to evaluate two TAC techniques in surgical and trauma patients requiring OAM: Barker's vacuum pack technique (BVPT) and the ABThera™ open abdomen negative pressure therapy system (NPWT). The two study groups of 280 patients were well matched in demographics. The 30-day fascial closure rate was 69% for NPWT and 51% for BVPT ($p = 0.03$). The 30-day all-cause mortality was 14% for NPWT and 30% for BVPT ($p = 0.01$). Multivariate logistic regression analysis identified that patients treated with NPWT were significantly more likely to survive than the BVPT patients [odds ratio 3.17 (95% confidence interval 1.22–8.26), $p = 0.02$]. Despite the positive message, this study failed to convince the scientific community that NPWT is superior, because of the heterogeneity of study patients.

Kirkpatrick et al. [67] investigated whether the ABThera™ device reduces systemic inflammation after abbreviated laparotomy. They conducted a single-center, randomized controlled trial. Forty-five adults with abdominal injury (46.7%) or intra-abdominal sepsis (52.3%) were randomly allocated to the ABThera ($n = 23$) or Barker's vacuum pack ($n = 22$). On study days 1, 2, 3, 7, and 28, blood and peritoneal fluid were analyzed for cytokines. The authors noted that there was a significantly lower peritoneal fluid drainage from the ABThera at 48 h after randomization. Despite this, there was no difference in plasma concentration of IL-6 at baseline versus 24 ($p = 0.52$) or 48 h ($p = 0.82$) between the groups. The cumulative incidence of fascial closure at 90 days was similar between groups. However, 90-day mortality was improved in the ABThera group (hazard ratio, 0.32; 95% confidence interval, 0.11–0.93; $p = 0.04$). They concluded that a survival difference was noted between patients randomized to the ABThera versus Barker's vacuum pack. However, the cause for this survival advantage was not clear.

The third study by Bruhin et al. [68] was a systematic review by an expert panel on fascial closure. Their conclusions: "The 72% fascial closure rate following use of commercial NPWT kits in OAM for non-septic patients was increased to 82% by the addition of a 'dynamic' closure method. Wittmann Patch (68%) and home-made NPWT (vac-pack) (58%) had lower closure rates.

Patients with septic complications achieved a lower rate of fascial closure than non-septic patients but NPWT with dynamic closure remained the best option to achieve fascial closure."

1.3 Other Important Landmarks in the History of OAM

Important landmarks in the history of OAM in this decade are the efforts on the part of major societies and organizations, both national and international, to standardize and quantify clinical variables, prognostic factors, and recommendations for future investigations. These range from the creation of databases, multi-institutional studies, practice management guidelines (PMG), etc. They are briefly enumerated here. The reader will undoubtedly find more details elsewhere in this book.

1.4 Standardization of Definitions and Grading the OAM

As mentioned earlier, the WSACS provided standardized definitions for the OAM and its related phenomena that are summarized in Table 1.3. Bjork et al., from the executive committee of the Society, graded the OAM in severity for comparing outcomes and amended it very recently [69]. It is as follows:

"Grade I, without adherence between the bowel and abdominal wall or fixity of the abdominal wall (lateralization), subdivided as 1A, clean; 1B, contaminated; and 1C, with enteric leak. An enteric leak controlled by closure, exteriorization into a stoma, or a permanent enterocutaneous fistula is considered clean.

Table 1.3 Consensus definitions and recommendations of the World Society of the Abdominal Compartment Syndrome

Definitions
1. IAP is the steady-state pressure concealed within the abdominal cavity
2. The reference standard for intermittent IAP measurements is via the bladder with a maximal instillation volume of 25 mL of sterile saline
3. IAP should be expressed in mmHg and measured at end-expiration in the supine position after ensuring that abdominal muscle contractions are absent and with the transducer zeroed at the level of the midaxillary line
4. IAP is approximately 5–7 mmHg in critically ill adults
5. IAH is defined by a sustained or repeated pathological elevation in IAP ≥ 12 mmHg
6. ACS is defined as a sustained IAP > 20 mmHg (with or without an APP <60 mmHg) that is associated with new organ dysfuncti on/failure
7. IAH is graded as follows
Grade I, IAP 12–15 mmHg; Grade II, IAP 16–20 mmHg; Grade III, IAP 21–25 mmHg; Grade IV, IAP > 25 mmHg

(continued)

Table 1.3 (continued)

Definitions
8. Primary IAH or ACS is a condition associated with injury or disease in the abdominopelvic region that frequently requires early surgical or interventional radiological intervention
9. Secondary IAH or ACS refers to conditions that do not originate from the abdominopelvic region
10.Recurrent IAH or ACS refers to the condition in which IAH or ACS redevelops following previous surgical or medical treatment of primary or secondary IAH or ACS
11.APP = MAP – IAP
Recommendations: *we recommend*
1. Measuring IAP when any known risk factor for IAH/ACS is present in a critically ill or injured patient [GRADE IC]
2. Studies should adopt the trans-bladder technique as the standard IAP measurement technique [not GRADED]
3. Use of protocolized monitoring and management of IAP versus not [GRADE IC]
4. Efforts and/or protocols to avoid sustained IAH as compared to inattention to IAP among critically ill or injured patients [GRADE IC]
5. Decompressive laparotomy in cases of overt ACS compared to strategies that do not use decompressive laparotomy in critically ill adults with ACS [GRADE ID]
6. Among ICU patients with open abdominal wounds, conscious and/or protocolized efforts be made to obtain an early or at least same-hospital-stay abdominal fascial closure [GRADE ID]
7. Among critically ill/injured patients with open abdominal wounds, strategies utilizing negative pressure wound therapy should be used versus not [GRADE IC]

From: Kirkpatrick A et al.: Intra-abdominal hypertension and the abdominal compartment syndrome: updated consensus definitions and clinical practice guidelines from the World Society of the Abdominal Compartment Syndrome. Intensive Care Med. 2013 Jul; 39(7): 1190–1206
ACS abdominal compartment syndrome, *IAP* intra-abdominal pressure, *IAH* intra-abdominal hypertension, *APP* abdominal perfusion pressure, *MAP* mean arterial pressure

Grade II, developing fixation, subdivided as follows: 2A, clean; 2B, contaminated; and 2C, with enteric leak.
Grade III, frozen abdomen, subdivided as follows: 3A clean and 3B contaminated.
Grade IV, an established enteroatmospheric fistula, defined as a permanent enteric leak into the open abdomen, associated with granulation tissue."

1.5 World Society of Emergency Surgery (WSES): International Registry of Open Abdomen (IROA)

Under the leadership of Federico Coccolini, the editor in chief of this book, this WSES initiative aims to overcome the lack of high level of evidence data about the OAM indications, management, definitive closure, and follow-up, "…warmly invite all surgeons or physicians who perform and manage with OAM procedures to participate to this international effort in order to get the best result and contribute to better understand the OAM procedure" [70].

1.6 OAM for Trauma: Recent Landmark Developments

1.6.1 EAST Practice Management Guidelines on OAM for Trauma

Eastern Association for the Surgery of Trauma (EAST) PMG Committee, as is customary to them, convened a study group to establish recommendations for the use of open abdomen techniques. The PMGs were published in three parts [71–73]: Part I "Damage Control," Part II "Management of the Open Abdomen," and Part III "Review of abdominal wall reconstruction." Each part provides a detailed literature review and discussion of specific topics of OAM, another landmark in the history of OAM.

1.6.2 AAST Open Abdomen Study Group on OAM for Trauma

Sponsored by the multi-institutional study committee and under the direction of DuBose, the "open abdomen study group" of AAST conducted a prospective observational multi-institutional study to examine the natural history of the open abdomen management (OAM) after trauma. They recruited 572 patients from 14 American College of Surgeons-verified Level I trauma centers. The overall mortality was 23%. Initial primary fascial closure (PFC) was achieved in 379 patients (66%). Development of intra-abdominal abscess/sepsis, acute renal failure, enteric fistula, and ISS > 15 were independent risk factors for failure of fascial closure [74]. Subsequent results from this group [75, 76] included predictors of enterocutaneous, enteroatmospheric fistulae and intra-abdominal sepsis. Delays in returning to the operating room after damage control laparotomy were associated with reduction in PFC. Severity of injury, plasma transfusions, and greater fluid administration by 24 h were independently associated with ARDS development.

1.6.3 International Consensus Conference on Open Abdomen in Trauma

Multiple leading international experts assembled to grade the evidence on OAM for trauma by grade of recommendation (GoR) and level of evidence (LoE) [77]:

- OAM in trauma is indicated at the end of damage-control laparotomy, in the presence of visceral swelling, for a second look in vascular injuries or gross contamination, in the case of abdominal wall loss, and if medical treatment of abdominal compartment syndrome has failed (GoR B, LoE II).
  - Negative-pressure wound therapy is the recommended temporary abdominal closure technique to drain peritoneal fluid, improve nursing, and prevent fascial retraction (GoR B, LoE I).

- Lack of OAM closure within 8 days (GoR C, LoE II), bowel injuries, high-volume replacement, and use of polypropylene mesh over the bowel (GoR C, LoE I) are risk factors for frozen abdomen and fistula formation.
- Negative-pressure wound therapy allows to isolate the fistula and protect the surrounding tissues from spillage until granulation (GoR C, LoE II).
- Correction of fistula is performed after 6–12 months. Definitive closure of OAM has to be obtained early with direct suture, traction devices, component separation with or without mesh (GoR C, LoE I).
- Biologic meshes are an option for wall reinforcement if bacterial contamination is present (GoR C, LoE II).

1.7 OAM for Abdominal Sepsis: Recent Landmark Developments

1.7.1 World Society of Emergency Surgery Position on OAM for Sepsis

OAM is a significant surgical advance also for patients with severe abdominal sepsis. It offers all the advantages as in DCS for trauma. "However, OAM for sepsis may be associated with significant complications. Surgeons should be aware of the pathophysiology of severe intra-abdominal sepsis and always keep in mind the option of using open abdomen to be able to use it in the right patient at the right time" [78].

1.7.2 Results of OAM for Abdominal Sepsis

It is also becoming widely accepted that OAM for trauma and abdominal sepsis have a variable course and different outcomes as the following most recent reports have documented.

A prospective observational study of emergency general surgery OAM patients (2013–2014) was published in April 2016 [79]. A total of 338 laparotomies were performed, of which 96 (28%) were managed with an OAM, with a median age of 61 years. The most common indications for operation were perforated viscus/free air (20%), mesenteric ischemia (17%), peritonitis (16%), and gastrointestinal hemorrhage (12%). DCS was the most common indication for OAM (37%). Sixty-three patients had fascial closure. Ten percent had fascial dehiscences. A total of 30% of the patients died in the hospital, and an additional six patients died 6 months after discharge. The authors concluded that older patients were more likely to die by 6 months and this group needed further study in OAM.

A recent report from Florida (October 2016) [80] reviewed OAM with TAC for trauma ($n = 77$) or intra-abdominal sepsis ($n = 147$). All patients received negative pressure wound therapy (NPWT) TAC with intention for planned relaparotomy and sequential abdominal closure attempts at 24–48-h intervals. Among patients surviving to discharge, trauma patients had higher primary fascial closure (PFC) rates (90

versus 76%). For trauma patients, independent predictors of failure to achieve PCF were ≥2.5 L NPWT output at 48 h, ≥10 L crystalloid administration at 48 h, and ≥10 U PRBC + FFP at 48 h. For septic patients, relaparotomy within 48 h predicted successful PFC; requirement for ≥3 diagnostic/therapeutic laparotomies predicted failure to achieve PFC.

The most recent report about OAM in November 2016 was a systematic review in accordance with PRISMA guidelines [81]. The objective of the study was to identify prognostic factors in OAM patients in regard to definitive fascial closure (DFC), mortality, and intra-abdominal complications. Thirty-one studies were included in the final synthesis. Enteral nutrition, organ dysfunction, local and systemic infection, number of re-explorations, worsening ISS, and the development of a fistula appeared to significantly delay DFC. Age and APACHE II score were predictors for in-hospital mortality. Failed DFC, large bowel resection, and >5–10 L of intravenous fluids in <48 h were predictors of enteroatmospheric fistula. Fascial closure on (or after) day 5 and having a bowel anastomosis were predictors for anastomotic leak. The authors noted the following limitations in the studies: only a moderate overall methodological quality, a high number of retrospective studies, and a low reporting of prognostic factors.

1.8 Current Status of Abdominal Defect Repair

The final frontier of OAM for sepsis in survivors, albeit still unconquered, is the delayed repair of abdominal wall defects and hernia. Here is the latest report on the subject [82]:

Thirty-two studies published between January 1990 and June 2015 on repair of (potentially) contaminated hernias with ≥25 patients were reviewed in this systematic analysis. Fifteen studies only described the use of biologic mesh, six nonabsorbable synthetic meshes, and 11 various techniques. Analysis showed no benefit of biologic over synthetic mesh. Biologic mesh repair had higher rates of surgical site complications and a hernia recurrence rate of 30%. (However, there was only one study on synthetic repair.) No definitive conclusions could be reached.

1.9 Summary

History has been defined as: “the continuum of events occurring in succession leading from the past to the present and even into the future” [83]. This chapter has attempted to trace OAM through its tortured path from birth to popularity through periods of skepticism and ridicule. As Thomas Starzl famously remarked: “New ideas seldom have the simplicity of a switched on light bulb” [83]. Even after the realization of the merits of the OAM, it had to go through periods of stunted development, waiting for the next good idea to take hold. Witness the fact that it took us almost a decade to realize the value of negative pressure and vacuum systems in OAM.

Conclusion

The open abdomen technique is one of the greatest advances in recent times and has enormous application in the daily management of the critically ill or injured patient. It results in tremendous benefits in the initial resuscitation of these patients. It also brings on many challenges beyond those that might be expected from the primary illness or injury. History has proven the value of this technique and taught us many lessons to overcome its challenges and reap the benefits. The future of OAM rests with better criteria to curtail misuse, higher level of evidence from controlled studies, and a critical evaluation of quality of life, both short and long term. The pace of progress in OAM, arguably, has quickened. Yet, much more remains to be accomplished. It will be. Surely.

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Basic Research in Open Abdomen

2

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2.1 Background

Basic research on open abdomen, especially using animal models, has recently developed around some important topics. The aim is to better understand this strategy and its pathophysiology and to allow its more effective use and prevent the complications of this procedure.

Actually the most studied topics in basic research are the immunological modifications caused by NPT (negative pressure therapy), the distribution of negative pressure in the abdominal cavity, its effects on bowel surface, and the development of means to protect intestinal anastomosis in the setting of open abdomen.

2.2 Immunological Modification

Basic research on open abdomen has focused on immunological modifications induced by NPT, not only in the peritoneal cavity but also in the systemic circulation. Recent preclinical studies have analyzed the role of the temporary abdominal closure systems that employ negative pressure in preventing multiple

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organ dysfunction syndrome (MODS) and in improving outcomes in animal models [1–4].

2.2.1 MODS Pathogenesis

MODS is the final and often lethal stage of the septic and hemorrhagic shock. The mechanism of MODS is thought to be the excessive systemic inflammation (SIRS, systemic inflammatory response syndrome) that affects a complex array of molecular pathways causing an overwhelming "cytokine storm" [1, 3].

Although there is no consensus about the organ system that generates MODS, it is well established that an injury in one organ system can cause injury to a second, distant organ system through the "cytokine storm" [5, 6].

It is likely that the gut is the initial motor of MODS, also during extra-abdominal sepsis. Indeed microcirculation in the gut is preferentially altered in both septic and hemorrhagic shock through hypoxic and inflammation-induced injury. This can lead to the loss of intestinal barrier function and to the increasing of permeability, resulting in intestinal edema and ascites formation. This fluid in the third space is rich in cytokines and inflammatory mediators that can reach the systemic circulation, perpetuate SIRS, and promote MODS [1].

Indeed it has been suggested by some studies that inflammatory mediators such as cytokines released during intestinal ischemia and reperfusion increase permeability in the lungs with subsequent lung injury [7]. To stress the important role of cytokines in causing distant organ system failure during MODS, in a study on animal models by Narita et al. [8], it has been demonstrated that the isolation of the intestine in a bag during ischemia and reperfusion reduced the degree of subsequent lung injury, probably due to the reduced absorption of locally produced cytokines via the parietal peritoneum [8].

So, in light of these studies, the local control of cytokines in the peritoneal cavity may be important in order to prevent MODS.

2.2.2 Preclinical Studies

In the study by Kubiak et al. [1], pigs with intra-abdominal sepsis were randomized to negative pressure peritoneal therapy versus passive drainage of the peritoneal cavity. The most important findings in this study were that peritoneal NPT reduced histologic damage to the lungs, intestine, kidney, and liver. The mechanism for this protection involved removal of inflammatory peritoneal ascites, causing a moderation of SIRS and a limitation of distant organ damage. The reduction in peritoneal inflammation was responsible for the blunted systemic inflammatory response in the NPT group, with plasma concentrations of TNF-α, IL 1-β, IL-6, and IL-12 diminished. Furthermore NPT, removing a larger volume of ascites, reduced intra-abdominal pressure (IAP), which may also play a role in the reduction of systemic inflammation and organ damage. These data suggest that inflammatory ascites, rather than bacterial translocation, is the motor driving organ damage.

Emr et al. [2] went further insight into the complex immunological response, through the use of "in vivo" and "in silico" studies. They found that only the concentrations of IL-8 and IL-6 were lowered significantly in NPT group, while all other inflammatory mediators remained at the same concentration. Given that IL-6 is the biomarker that typically distinguishes adverse outcomes in sepsis [9], probably NPT modifies inflammation in a favorable fashion by reducing the production of IL-6. There were differences neither in endotoxin concentration, and all animals had positive cultures for both aerobic and anaerobic bacteria. Thus, the simple removal of bacteria does not seem to be the mechanism underlying the protective effect of NPT.

Furthermore, in silico analysis, IL-10, which is inferred to be produced as a consequence of the core IL-6/TGF-β1/CRP motif in control group, is absent in NPT group. IL-10 is a key anti-inflammatory cytokine, so its absence suggests a more robust ability to control infection with NPT.

Norbury et al. [3] in 2015 developed these aspects analyzing in more details the immunological modifications induced by NPT in the septic swine model. Also in this study, the improved survival due to NPT was directly associated with a reduction of MODS. Analyzing the immunological response, in both groups (peritoneal NPT versus passive peritoneal drainage), the septic swine model displayed evidence of leukocytosis in the initial 12 h after injury, followed by immunoparalysis manifested as lymphopenia. This is the result of a compensatory anti-inflammatory response designed to protect against an uncontrolled hyperinflammatory response. While in the group treated with passive drainage there was a further decrease in the number of circulating lymphocytes as the experiment progressed, the NPT group showed a significant recovery by the end of the experiment, suggesting that NPT mitigated the further effects of systemic inflammatory injury and overcome the effects of immunoparalysis in this model.

In the NPT group also, the response by macrophages in producing ROS was demonstrably greater and peaked early with the effect being even greater at 3 h than at 6 h, again suggesting that the inflammatory response is more effective but finite and controlled.

So this study hypothesized that the effect of NPT on the inflammatory response is not only due to the active removal of inflammatory mediators from the peritoneal cavity before they enter in the systemic circulation but also due to a dynamic alteration of the microenvironment that allows a more robust, yet transient, innate antimicrobial response.

Regarding trauma setting, a recent study by Shi et al. [4] showed that in experimental pigs with blast injury in the abdomen and exposed internal organ, NPT can control the amount of bacteria; reduce the expression of TNF-α, IL-1, and IL-6; and promote the expression of growth factors.

In a study by Kuethe et al. [9], peritoneal cytokines and cells of mice with abdominal sepsis were analyzed. Mice that survived had decreased peritoneal IL-6 levels, decreased peritoneal bacterial loads, decreased systemic IL-10, and increased peritoneal monocyte numbers and phagocytosis. All these are the observed effects of NPT in the septic swine model in the previously mentioned studies.

2.3 Distribution of Negative Pressure in the Abdominal Cavity and Its Effects on Bowel Surface

Some preclinical studies focused on pressure distribution in abdominal cavity and its effects on microvascular blood flow in the intestinal wall using different open abdomen dressing systems [10–15].

2.3.1 Pressure Distribution

In a recent in vitro study by Delgado et al. [10], assessing pressure mapping and fluid extraction efficiency of three open abdomen dressing systems (ABThera™ Active Abdominal Therapy System, VAC® Abdominal Dressing System, and Barker's vacuum pack technique), pressure distribution of ABThera Therapy and of VAC Abdominal Dressing System was significantly superior to Barker's vacuum pack technique in all peritoneal evaluated zones. There were no pressure distribution differences in the zone closest to negative pressure source between ABThera Therapy and VAC Abdominal Dressing System. In the zone immediately outside of manifolding material edge and in the area most distant to negative pressure source, ABThera Therapy pressure was significantly superior to VAC Abdominal Dressing System one.

2.3.2 NPT Effects on Intestinal Wall

In the study by Bjarnason [11], pressure propagation at the bowel surface was investigated on a porcine model. The negative pressure observed at the bowel surface was substantially reduced, compared to the applied pressure, for all NPT settings, indicating that the visceral protective layer effectively isolates the bowel from the negative pressure. Furthermore in this study, the observed pressure at the bowel surface did not correlate with the level of applied NPT.

Lindstedt et al. conducted many studies on pigs about pressure transduction on intestinal wall, fluid evacuation, and protection of intestinal loops during open abdomen. They showed that the use of NPT in open abdomen induces a decrease in the blood flow in the small intestinal wall lying close to the dressing and beneath the anterior abdominal wall. Furthermore, in spite of Bjarnason's results, the decrease in blood flow became greater with increasing negative pressure applied. The blood flow could be restored by inserting a protective thin plastic disc over the intestine but could not be prevented by inserting four layers of paraffin gauze between the visceral protective layer and the intestine [12, 13]. Macroscopic changes in the small intestine lying close to the NPT dressing were studied in another preclinical trial [14]. Slight petechial bleeding was seen in the small intestinal loop in this area after 24 h, but especially after 48 h. In contrast, hardly any petechial bleeding was seen in pigs treated with the protective disc over the intestines [14]. Furthermore, abdominal drainage was significantly better using NPT with the protective disc than

with conventional NPT system, and the pressure transduction was more even at all pressure levels using NPT with protective disc than with conventional NPT [13].

Comparing VAC Abdominal Dressing System and ABThera Therapy, there were no differences in the decrease in microvascular blood flow in the intestinal wall lying close to the visceral protective layer. So the decrease in the blood flow was related to the amount of negative pressure applied and not to the type of dressing [15]. These results are very interesting if applied to humans, and the use of the protective plastic disc over the intestine could be a useful tool to prevent intestinal injury in these patients.

2.4 Means to Protect Intestinal Anastomosis in Open Abdomen

Zhou et al. conducted a study to evaluate the effect of platelet-rich plasma (PRP) gel, a blood-derived biomaterial, on the healing of colon anastomosis and anastomotic strength in the open abdomen [16]. There is a growing body of evidence indicating that blood-derived biomaterials such as platelet-rich plasma gel promote wound healing in a variety of clinical fields via release of chemoattractants and growth factor, such as platelet-derived growth factor (PDGF) and transforming growth factor-β (TGF-β). These molecules synergize to facilitate regeneration of injured tissue through acceleration of cell proliferation and matrix formation [17]. They observed that the hydroxyproline levels (amino acid found almost exclusively in collagen, used as an indicator of collagen synthesis and wound healing) were statistically higher in patients with open abdomen that were treated with PRP on anastomosis than in patients with traditional open abdomen. They analyzed the anastomotic bursting pressure that reflects the balance between collagen deposition and lysis, and it's a predictor of outcome of gastrointestinal anastomoses. In anastomoses in open abdomen patients treated with PRP, after day 7, the bursting pressure was similar to anastomoses in patients without open abdomen and higher than in patients treated with traditional open abdomen technique without PRP. Furthermore, colonic anastomoses sealed by PRP gel showed significantly increased inflammatory cell infiltration, anastomotic fibroblast ingrowth, anastomotic neovascularization, and collagen deposition compared to standard anastomoses [16].

Other substances were tested in other studies with the same aim. In a study in rat models, tannin acid–polyethylene glycol adhesive seems to give good results in this context [18].

Conclusion

Basic research on open abdomen and NPT is in constant evolution.

NPT in septic animal models seems to offer better outcomes in terms of mortality and morbidity. This advantage can be explained by the local and systemic immunologic modification caused by removing abdominal fluids rich in inflammatory mediators. This results in more effective and more limited local inflammatory response, in lower levels of circulating cytokines, and in decreased risk

of distant organ injury and MODS. Evidence obtained from animal models can potentially be applied to clinical research, thus providing new insight in the pathophysiological basis of NPT.

Basic research also focuses on complications of negative pressure on intestinal wall and anastomotic leaks. Recent reports have shown promising results on how to prevent those complications.

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Anatomy and Physiology of the Abdominal Compartment

3

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3.1 Introduction

Over the last decades, increasing attention has been paid understanding the importance of intra-abdominal pressure (IAP), intra-abdominal hypertension (IAH), and the abdominal compartment syndrome (ACS) [1–6]. While IAH is a graded phenomenon, ACS is an all-or-none condition causing dysfunction of neurological, respiratory, cardiovascular, renal, and hepatic organ systems [7]. This chapter provides a detailed overview of the historical background, definitions, classification, the different IAP measurement techniques, epidemiology, and pathophysiology of IAH and ACS.

3.2 Historical Background

The historical background surrounding IAH and ACS is nicely summarized in an excellent review paper [8]. Paul Bert (1833–1886) measured pressures through tubes inserted in the trachea and rectum in France, in Claude Bernard's laboratory. He described elevation of IAP during inspiration due to diaphragmatic descent. Marey was the first to report that the "effects that respiration produces on the thorax are the inverse of those present in the abdomen." Wendt later reported an inverse

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relationship between rectal IAP and urinary secretion. In 1890, Henricus reported that severe IAH impaired respiration, decreased cardiac diastolic distention, and induced hypotension.

The first intravesicular or bladder pressure measurements were performed by the Italians Mosso and Pellacani in 1881. Direct puncture of the peritoneal cavity is another direct method of IAP measurement; however, its use cannot be advocated in critically ill. Once the nineteenth century era of pressure measurements came along, IAP was evaluated in different physiological and pathological circumstances. In 1911, Haven Emerson (1874–1957) published his epoch-making results of IAP measurements [9]. He demonstrated that IAH impaired cardiac venous return, increased systemic vascular resistance, decreased cardiac contractility and arterial blood pressure, and could cause death from cardiac failure.

3.3 Definitions

The WSACS (the Abdominal Compartment Society, formerly known as the World Society of the Abdominal Compartment Syndrome [10]) has published guidelines for definition, diagnosis, and management of IAH/ACS starting in 2006 and 2007 [1, 2], and these have been recently revised according to the GRADE methodology [3]. The latest WSACS consensus definitions are listed in Table 3.1.

3.3.1 The Abdominal Compartment

The abdominal compartment is a technical masterpiece as the small abdominal cavity houses 8.5 m of intestine [11, 12]. Analogous to the head, the abdomen may be considered a closed box with an anchorage above (costal arch) and rigid (spine and pelvis) or partially flexible sides (abdominal wall and diaphragm) filled with large vessels, intestines, and organs. The size and/or volume of the abdomen may be affected by the varying location of the diaphragm, the shifting position of the costal arch, the contractions of the abdominal wall, the filling status, and the amount of contents (air, liquid, feces, or even blood) contained within the intestines. The caudal and dorsal parts of the abdomen are rigid structures formed by the pelvic bones and dorsal spine. Only the ventral (abdominal wall and muscles) and cranial (diaphragm) parts of the abdominal cavity are relatively flexible [13, 14].

3.3.2 The Abdominal Wall

The abdominal wall represents the boundaries of the abdominal cavity between the xyphoid bone and costal margins cranially and the iliac and pubis bones of the pelvis caudally [15]. There is a common set of layers covering and forming the abdominal wall: the deepest are the extraperitoneal fat and peritoneum. Superficial to these is the rectus abdominis muscle and its associated fascia. The abdominal muscles

Table 3.1 WSACS consensus definitions regarding intra-abdominal hypertension (IAH) and abdominal compartment syndrome (ACS) according to the 2006 and 2013 WSACS guidelines update (adapted from Malbrain et al. [1] and Kirkpatrick et al. [3])

Def	2006 definitions [1]	Def	2013 definitions [3]
1	IAP is the steady-state pressure concealed within the abdominal cavity	1	IAP is the steady-state pressure concealed within the abdominal cavity
2	APP = MAP – IAP	2	APP = MAP – IAP
3	FG = GFP – PTP = MAP – 2 × IAP		REJECTED
4	IAP should be expressed in mmHg and measured at end-expiration in the complete supine position after ensuring that abdominal muscle contractions are absent and with the transducer zeroed at the level of the midaxillary line	3	IAP should be expressed in mmHg and measured at end-expiration in the complete supine position after ensuring that abdominal muscle contractions are absent and with the transducer zeroed at the level of the midaxillary line
5	The reference standard for intermittent IAP measurement is via the bladder with a maximal instillation volume of 25 mL of sterile saline	4	The reference standard for intermittent IAP measurements is via the bladder with a maximal instillation volume of 25 mL of sterile saline
6	Normal IAP is approximately 5–7 mmHg in critically ill adults	5	IAP is approximately 5–7 mmHg and around 10 mmHg in critically ill adults
7	IAH is defined by a sustained or repeated pathologic elevation of IAP ≥ 12 mmHg	6	IAH is defined by a sustained or repeated pathologic elevation of IAP ≥ 12 mmHg
8	IAH is graded as follows • Grade I: IAP 12–15 mmHg • Grade II: IAP 16–20 mmHg • Grade III: IAP 21–25 mmHg • Grade IV: IAP > 25 mmHg	7	IAH is graded as follows • Grade I: IAP 12–15 mmHg • Grade II: IAP 16–20 mmHg • Grade III: IAP 21–25 mmHg • Grade IV: IAP > 25 mmHg
9	ACS is defined as a sustained IAP ≥ 20 mmHg (with or without an APP < 60 mmHg) that is associated with new organ dysfunction/failure	8	ACS is defined as a sustained IAP ≥ 20 mmHg (with or without an APP < 60 mmHg) that is associated with new organ dysfunction/failure
10	Primary ACS is a condition associated with injury or disease in the abdominopelvic region that frequently requires early surgical or interventional radiological intervention	9	Primary ACS is a condition associated with injury or disease in the abdominopelvic region that frequently requires early surgical or interventional radiological intervention
11	Secondary ACS refers to conditions that do not originate from the abdominopelvic region	10	Secondary ACS refers to conditions that do not originate from the abdominopelvic region
12	Recurrent ACS refers to the condition in which ACS redevelops following previous surgical or medical treatment of primary or secondary ACS	11	Recurrent ACS refers to the condition in which ACS redevelops following previous surgical or medical treatment of primary or secondary ACS

(continued)

Table 3.1 (continued)

Def	2006 definitions [1]	Def	2013 definitions [3]
		12	**New**: A polycompartment syndrome is a condition where two or more anatomical compartments have elevated compartmental pressures
		13	**New**: Abdominal compliance quantifies the ease of abdominal expansion, is determined by the elasticity of the abdominal wall and diaphragm, and is expressed as the change in intra-abdominal volume per change in intra-abdominal pressure in L/mmHg
		14	**New**: An open abdomen (OA) is any abdomen requiring a temporary abdominal closure due to the skin and fascia not being closed after laparotomy. The technique of temporary abdominal closure should be explicitly described
		15	**New**: The open abdomen is classified with the following grading system **1—no fixation** 1A: clean, no fixation 1B: contaminated, no fixation 1C: enteric leak, no fixation **2—developing fixation** 2A: clean, developing fixation 2B: contaminated, developing fixation 2C: enteroatmospheric/cutaneous fistula, developing fixation **3 and 4—frozen abdomen** 3: frozen abdomen, no fistula 4: frozen abdomen with enteroatmospheric/cutaneous fistula
		16	**New**: Lateralization of the abdominal wall refers to the phenomenon whereby the musculature and fascia of the abdominal wall, most well seen by the rectus abdominis muscles and their enveloping fascia, move laterally away from the midline with time

ACS abdominal compartment syndrome, *FG* filtration gradient, *GFP* glomerular filtration pressure, *IAH* intra-abdominal hypertension, *IAP* intra-abdominal pressure, *MAP* mean arterial pressure, *OA* open abdomen, *PTP* proximal tubular pressure

have a composite-laminated structure determining their nonlinear stretch capacities [16]. The *m. transversus abdominis* fascial fibers are responsible for the transverse stiffness of the abdominal wall, whereas the *m. rectus abdominis* is much more compliant in the sagittal plane [13].

3.3.3 Intra-abdominal Pressure

Intra-Abdominal Pressure The IAP is the steady-state pressure concealed within the abdominal cavity [3].

Baseline IAP The baseline IAP depends on the amount of "prefilling" of the abdominal cavity in proportion to the compliance of the abdominal wall and status or contractions of the diaphragm. Normal IAP is approximately 5–7 mmHg in healthy individuals, 4–10 mmHg in critically ill children, and around 10 mmHg or above in critically ill adults. Baseline IAP is also increased in obese patients, during pregnancy, in patients undergoing chronic ambulatory peritoneal dialysis, and in the postoperative period (after laparotomy).

Intra-abdominal Hypertension IAH is defined by a sustained or repeated pathological elevation in IAP $\geq$ 12 mmHg.

IAH Grading IAH is graded as follows: Grade I, IAP 12–15 mmHg; Grade II, IAP 16–20 mmHg; Grade III, IAP 21–25 mmHg; and Grade IV, IAP > 25 mmHg.

Abdominal Compartment Syndrome ACS is defined as a sustained IAP > 20 mmHg (with or without an abdominal perfusion pressure (APP) < 60 mmHg) that is associated with new organ dysfunction/failure. In contrast to IAH, ACS is an all-or-nothing phenomenon.

Delta IAP ΔIAP is calculated as the difference between the end-inspiratory IAP (IAP_{ei}) and the end-expiratory IAP (IAP_{ee}) value, $\Delta IAP = IAP_{ei} - IAP_{ee}$. The higher the ΔIAP, the lower the abdominal compliance (C_{ab}) [17].

Abdominal Pressure Variation APV is calculated as the difference between the end-inspiratory IAP (IAP_{ei}) and the end-expiratory IAP (IAP_{ee}) value (or thus ΔIAP) divided by the mean IAP (MIAP) and expressed as a percentage, $APV = \Delta IAP/MIAP = (IAP_{ei} - IAP_{ee})/MIAP$. The higher the APV, the lower the abdominal compliance (C_{ab}) [17].

3.3.4 Intra-abdominal Volume (IAV)

Baseline IAV This is also called resting or static IAV which is the IAV at baseline conditions without additional pathologic volume increase or C_{ab} decrease, with a corresponding baseline IAP [12]. The baseline IAV in healthy individuals is around 10–13 L [18].

Abdominal Distention This is defined as a sagittal abdominal diameter (approximately at the level of the umbilicus) higher than the virtual line between xiphoid and symphysis pubis.

Abdominal Workspace This is the additional IAV that can be added upon the baseline IAV when IAP is limited to a certain pressure (e.g., 14 mmHg during laparoscopic surgery) [12]. The normal workspace during laparoscopy ranges between 3 and 6 L [19, 20].

Maximal Stretched Volume The maximal volume is calculated as the baseline IAV plus the maximal workspace resulting in maximal stretch of the abdominal cavity (changing from ellipse to sphere shaped on transverse plane) [12].

3.3.5 Abdominal Compliance (C_{ab})

Abdominal Compliance Abdominal compliance (C_{ab}) is defined as a measure of the ease of abdominal expansion, which is determined by the elasticity of the abdominal wall and diaphragm [3]. An increased compliance indicates a loss of elastic recoil of the abdominal wall (e.g., as seen in old age). A decreased compliance means that the same change in IAV will result in a greater change in IAP (as seen in young persons with increased abdominal muscles, e.g., body builders). The C_{ab} should be expressed as the change in intra-abdominal volume (IAV) per change in IAP (mL/mmHg). Normal C_{ab} is around 250–450 mL/mmHg [11].

Abdominal PV Relationship The relation between pressure and volume can be expressed by the analysis of PV curves (plotting the resulting changes in IAP values with corresponding changes in IAV, e.g., during laparoscopy), similar to the intracranial compartment. The abdominal compliance is calculated by the change in volume over the change in pressure or thus [21]:

$$C = \frac{\Delta V}{\Delta P} \text{ or thus } C_{ab} = \frac{\Delta IAV}{\Delta IAP}$$

The relation between abdominal volume and abdominal pressure is curvilinear with an initial linear part followed by an exponential increase once a critical volume is reached [12].

3.3.6 Classification

IAH and ACS may be classified as primary, secondary, or recurrent [3].

Primary IAH/ACS (formerly termed surgical or abdominal IAH/ACS) is "a condition associated with injury or disease in the abdominopelvic region that may require surgical or radiological intervention or stabilization." Common examples of conditions leading to primary ACS include severe acute pancreatitis, abdominal trauma (with spleen or liver rupture), or ruptured abdominal aortic aneurysms.

Secondary IAH/ACS (formerly termed medical or extra-abdominal IAH/ACS) refers to "a condition that does not originate in the abdominopelvic region." Cases with secondary ACS typically develop as a result of a capillary leak syndrome (secondary to sepsis, infection or inflammation, major burns, or other causes) and other conditions requiring massive fluid resuscitation (e.g., vascular extremity injury).

Recurrent IAH/ACS (previously termed tertiary IAH/ACS) is characterized by redevelopment of IAH/ACS following surgical (e.g., decompressive laparotomy) or medical minimally invasive (e.g., percutaneous catheter drainage) treatment of IAH/ACS.

3.4 Risk Factors Related to Abdominal Hypertension

The C_{ab} is one of the most neglected parameters in critically ill patients; however, it plays a very important role in understanding the deleterious effects of unadapted IAV on IAP and end-organ perfusion [17]. Measurement of C_{ab} is difficult at the bedside and can only be done in case of a known change (removal or addition) in IAV. Nevertheless, some indirect measures of C_{ab} are available in critically ill patients that are mechanically ventilated: the ΔIAP (= IAP at the end of inspiration minus the IAP at the end of expiration) and the abdominal pressure variation (APV = ΔIAP divided by mean IAP) are such parameters, and they are inversely correlated with C_{ab}, i.e., the higher ΔIAP or APV, the lower C_{ab}. As stated, true C_{ab} can only be measured in case of addition or removal of a known abdominal volume (e.g., during laparoscopic insufflation, paracentesis, gastric aspiration, etc.) with simultaneous measurement of the change in IAP. Therefore, if we could identify patients with a low C_{ab}, we could anticipate and select the most appropriate surgical treatment in order to avoid possible complications related to IAH/ACS. Theoretically C_{ab} not only allows prediction of complications during laparoscopy or mechanical ventilation, but it also allows identification of patients that would benefit from leaving their abdomen open, those in whom to monitor IAP, or those at risk during prone ventilation, etc. The risk factors for IAH and ACS can be classified into those decreasing abdominal compliance, those increasing intra-abdominal or intraluminal contents, and those related to capillary leak and fluid overload [22]. Table 3.2 lists some common conditions related to increased IAP. Aside from risk factors for IAH, patients should also be screened for risk factors for decreased C_{ab} (Table 3.3).

Table 3.2 Risk factors associated with increased IAP (adapted from Malbrain et al. [11] with permission)

A. Related to increased intra-abdominal contents
– Gastroparesis
– Gastric distention
– Ileus
– Volvulus
– Colonic pseudo-obstruction
– Abdominal tumor
– Retroperitoneal/ abdominal wall hematoma
– Enteral feeding
– Intra-abdominal or retroperitoneal tumor
– Damage control laparotomy
B. Related to abdominal collections of fluid, air, or blood
– Liver dysfunction with ascites
– Abdominal infection (pancreatitis, peritonitis, abscess, etc.)
– Hemoperitoneum
– Pneumoperitoneum
– Laparoscopy with excessive inflation pressures
– Major trauma
– Peritoneal dialysis
C. Related to capillary leak and fluid resuscitation
– Acidosis[a] (pH below 7.2)
– Hypothermia[a] (core temperature below 33°C)
– Coagulopathy[a] (platelet count below 50,000/mm^3 OR an activated partial thromboplastin time (APTT) more than two times normal OR a prothrombin time (PTT) below 50% OR an international standardized ratio (INR) more than 1.5)
– Polytransfusion/trauma (>10 units of packed red cells/24 h)
– Sepsis (as defined by the American–European Consensus Conference definitions)
– Severe sepsis or bacteremia
– Septic shock
– Massive fluid resuscitation (>5 L of colloid or >10 L of crystalloid/24 h with capillary leak and positive fluid balance)
– Major burns

[a]The combination of acidosis, hypothermia, and coagulopathy has been forwarded in the literature as the deadly triad [23, 24]

3.5 Epidemiology

The reported incidence of IAH ranges between 21 and 58%, while for ACS it ranges between 1 and 12% among general ICU patients [4–6]. Potential explanations for heterogeneity in reported incidence and prevalence estimates across studies include differences in recruited patient samples, differences in IAH grading and classification, and differences in IAP measurement techniques. A recent individual patient data meta-analysis of 1669 adult general ICU patients reported that 27.7% had IAH and 2.7% had ACS (as defined by the WSACS) at ICU admission [6]. When compared to mixed populations of adult ICU patients, trauma and emergency general surgery patients and those with ruptured AAAs, severe acute pancreatitis, and burns appear to have a substantially higher incidence and prevalence of IAH and ACS [25, 26]. In a study by Vidal and colleagues, 53% of trauma and emergency surgery

Table 3.3 Factors associated with decreased abdominal compliance (adapted from Malbrain et al. [11] with permission)

A. Related to anthropomorphy and demographics
• Male gender
• Young age (good elastic recoil)
• Obesity (weight, BMI)
• Android composition (sphere, apple shape)
• Increased visceral fat
• Waist-to-hip ratio > 1
• Short stature
B. Related to comorbidities and/or increased noncompressible IAV
• Fluid overload
• Bowels filled with fluid
• Stomach filled with fluid
• Tense ascites
• Hepatomegaly
• Splenomegaly
• Abdominal fluid collections, pseudocyst, abscess
• Sepsis, burns, trauma, and bleeding (coagulopathy)
C. Related to abdominal wall and diaphragm
• Umbilical hernia repair
• Muscle contractions (pain)
• Body builders (six pack)
• Interstitial and anasarca edema (skin, abdominal wall)
• Abdominal burn eschars (circular)
• Thoracic burn eschars (circular)
• Tight closure after abdominal surgery
• Abdominal Velcro belt or adhesive drapes
• Prone positioning and prone ventilation
• HOB > 45°
• Pneumoperitoneum
• Pneumatic anti-shock garments
• Abdominal wall bleeding
• Rectus sheath hematoma
• Correction of large hernias
• Gastroschisis
• Omphalocele
• Mechanical ventilation (positive pressure)
• Fighting with the ventilator
• Use of accessory muscles
• Use of positive end-expiratory pressure (PEEP)
• Presence of auto-PEEP (tension pneumothorax)
• COPD emphysema (diaphragm flattening)
• Basal pleuropneumonia

patients developed IAH, while more than 12% developed ACS during their ICU stay [27]. Another study of patients with major torso trauma-related hemorrhagic shock requiring massive fluid resuscitation reported a 13% incidence of ACS [28]. The

incidence of IAH/ACS among patients with burns is correlated with the volume of crystalloids infused during resuscitation and the total percentage of burned surface area (TBSA) [29, 30]. Even in severe burn patients without abdominal injury or surgery or even in those without abdominal wall burn eschars, IAH and ACS can still develop during the course of the disease [29, 30]. Patients with very extensive burns above 50% TBSA or higher are most likely to develop IAH/ACS, particularly if they have concurrent inhalational injury [26].

3.6 Abdominal Pressure Measurement

The WSACS recommends measuring IAP "when any known risk factor for IAH/ACS is present in a critically ill or injured patient" [3]. Because of the fluidlike nature of the abdomen following Pascal's law, the IAP can be measured in nearly every body part: rectal, uterine, inferior vena cava, femoral vein, bladder, and gastric pressure measurements have all been described and suggested as IAP surrogates or estimates, one even better than the other [31]. As stated before, the use of direct intraperitoneal pressure measurement cannot be advocated in patients because of the complication risks (bleeding, infection) and should only be used in an experimental setting or when combined with fluid drainage (paracentesis). A recent study concluded that femoral vein pressure (FVP) cannot be recommended as a surrogate measure for IAP even at IAP values above 20 mmHg. However, an elevated FVP was a good predictor of IAH. Over the years, bladder pressure measurements have been forwarded as the gold standard technique [3]. Figure 3.1 shows a homemade device for repeated IAP measurements via the bladder. As shown in Fig. 3.2, intermittent screening for IAH can be performed by measuring the height of the urine column as a cost-effective estimate for IAP (with the FoleyManometer, Holtech Medical, Charlottenlund, Denmark or the UnoMeter AbdoPressure, Convatec, Greensboro, North Carolina, USA) [33].

3.7 Pathophysiologic Implications of Increased IAP

Being linked and bound by the diaphragm, the thoracic and abdominal compartment cannot be seen in isolation. As stated above, Haven Emerson conducted numerous experiments in dogs showing that the contraction of the diaphragm is the main factor causing the rise in IAP during inspiration [9]. Changes in IAP are paralleled by changes in pleural pressures. Changes in thoracic compliance are reflected by changes in abdominal compliance and vice versa; as a consequence, increased IAP will result in reduced chest wall compliance. The interactions between different body compartments have been referred to as the polycompartment model and syndrome [7, 34]. For instance, transmission of airway pressures to the abdomen results from interactions between the thoracic and abdominal compartment, and the percentage of pressure transmission is called the thoracoabdominal index (TAI) of transmission. This occurs in patients under positive pressure ventilation, application

of positive end-expiratory pressure (PEEP), presence of intrinsic or auto-PEEP, or a tension pneumothorax. Conversely transmission of pressure from the abdomen to the thorax is called abdomino-thoracic index of transmission (ATI), and this occurs in any physiologic (pregnancy) or pathologic condition associated with increased IAP. The ATI ranges from 20 to 80% and is on average 50% [35]. The knowledge of these interactions help us to understand the cardiorespiratory dynamics of patients with IAH/ACS. As a rule of thumb, transmural CVP (CVP_{tm}) can be calculated as follows based on the end-expiratory value (CVP_{ee}): $CVP_{tm} = CVP_{ee} - (IAP \times ATI)$. The interactions between compartments are not only dependent on the specific elastance of the different components but also on baseline pressures within the different compartments. Increased IAP has a two-sided effect: the abdominal wall is moved outward (abdominal extension), and the gaseous contents of hollow organs within the abdominal cavity are compressed inward since gas is compressible, while fluid

Set-Up:

- Wash hands and follow universal antiseptic precautions
- A Foley catheter is placed under sterile conditions and the urinary drainage system connected.
- Using a sterile field and gloves, the drainage tubing is cut (with sterile scissors) 40cm after the culture aspiration port after desinfection.
- A ramp with 3 stopcocks is connected to a conical connection piece at each side with a male/male adaptor.
- The ramp is then inserted in the drainage tubing.
- A standard intravenous (IV) infusion set is connected to a bag of 500mL of normal saline or D5W and attached to the first stopcock.
- A 60-mL syringe is connected to the second stopcock and the third stopcock is connected to a pressure transducer via rigid pressure tubing.
- The system is flushed with normal saline
- The pressure transducer is fixed at the symphysis or the thigh.
- Connect the transducer to the monitor via the special pressure module and ensure a normal waveform on the scope.
- Select a scale from 0 to 20 or 40 mmHg

Method of measurement:

- If the patient is awake, explain the procedure.
- If the patient is sedated, ensure good sedation.
- Place the patient in a complete supine position.
- Zero the pressure module at the midaxillary line of the patient at the level of the iliac crest (mark for future reference) by turning the proximal stopcock on to the air and the transducer
- At rest the 3 stopcocks are turned “off” to the IV bag, the syringe and transducer giving an open way for urine to flow into the urometer or drainage bag, said otherwise the 3 stopcocks are turned “on” to the patient.
- To measure IBP, the urinary drainage tubing is clamped distal to the ramp-device and the third stopcock is turned “on” to the transducer and the patient and “off” to the drainage system.
- The third stopcock also acts as a clamp.
- The first stopcock is turned “off” to the patient and “on” to the IV infusion bag, the second stopcock is turned “on” to the IV bag and the 60-mL syringe.
- Aspirate 20-25ml of normal saline from the IV bag into the syringe.
- The first stopcock is turned “on” to the patient and “off” to the IV bag and the 20-25ml of normal saline is instilled in the bladder through the urinary catheter.
- The first and second stopcock are then turned “on” to the patient, and thus turned “off” to IV tubing and the syringe.
- The third stopcock already being turned “on” to the transducer and patient allows then immediate IBP reading on the monitor.

Fig. 3.1 Modified method for intrabladder pressure monitoring (adapted from Desie et al. [32] with permission)

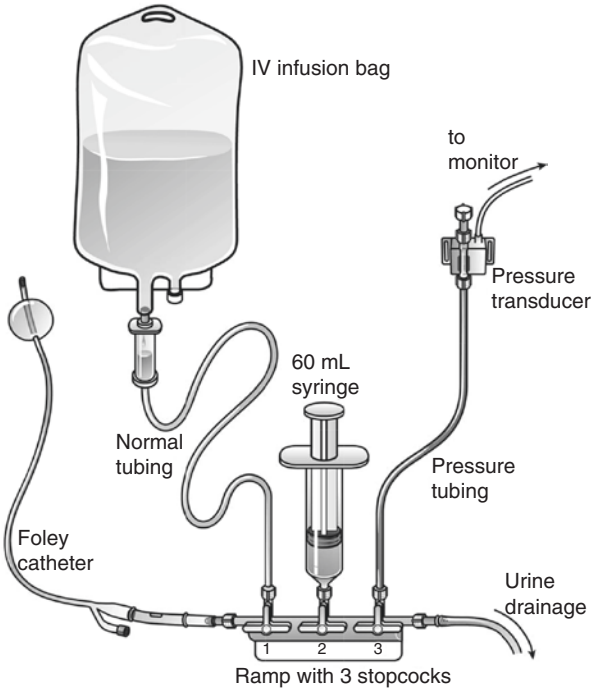


Fig. 3.1 (continued)

is not. The effects of increased IAP on end-organ function are numerous: neurologic, respiratory, cardiovascular, hepatic, and renal adverse effects have all been described in patients with IAH and ACS [7, 36–39]. Increased IAP leads to organ dysfunction of all abdominal organs due to arterial blood flow limitation, venous outflow obstruction (diminished venous return to the heart), and impaired microcirculatory flow, necessitating more fluid loading, causing mesenteric vein compression and venous hypertension, and finally triggering a vicious cycle. Kidney failure is the most often and consistently described organ failure associated with IAH, but hepatic, adrenal, and gastrointestinal dysfunctions have also been reported repeatedly. It is especially important to note that IAH can lead to increased bacterial translocation lending truth to the statement "the gut is the motor of sepsis." Figure 3.3 illustrates the different deleterious effects of increased IAP on end-organ function.

This technique that uses the patient's own urine as pressure transmitting medium is a surprisingly simple, reliable, and cost-effective clinical tool. Based on a modified version of the IAP monitoring technique described by Kron et al. (40), the disposable FoleyManometer (or UnoMeter AbdoPressure) provides a closed sterile circuit which connects between the patient´s Foley catheter and the urine collection device. Each IAP determination takes about 10 seconds, and no subsequent correction of urine output is required. The technique uses a low bladder infusion volume, has a needle-free sampling port and can measure IAP in a range from 0 - 40 mmHg. Therefore, it is an ideal technique to screen critically ill patients for IAH.

PANEL A, Initial set-up:

- Open the FoleyManometer LV (Holtech Medical, Charlottenlund, Denmark, www.holtech-medical.com) or UnoMeter AbdoPressure (Convatec, Greensboro, North Carolina, USA) pouch and close the tube clamp
- Place the urine collection device under the patient's bladder and tape the drainage tube to the bed sheet.
- Insert the FoleyManometer between catheter and drainage device.
- Prime the FoleyManometer with 20ml of sterile saline through its needle-free injection/sampling port.
- Prime only once i.e. at initial set-up, or subsequently to remove any air in the manometer tube.

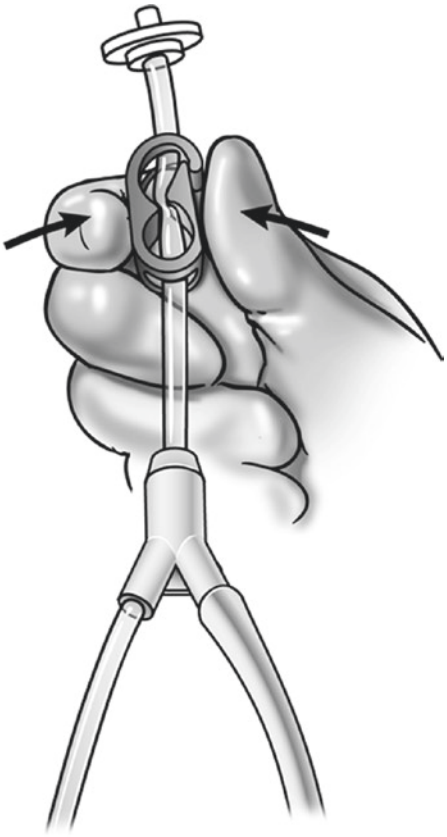
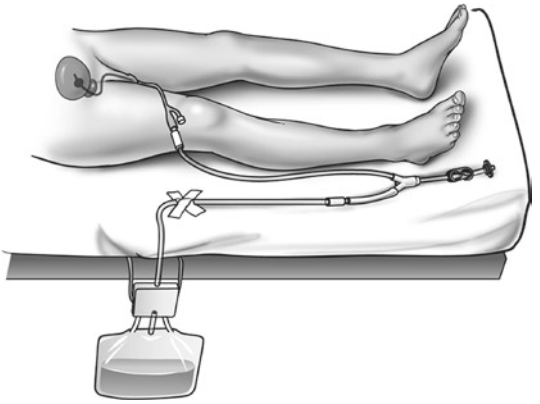


Fig. 3.2 Intrabladder pressure monitoring with the FoleyManometerLV (adapted from Desie et al. [32] with permission)

PANEL B, Urine drainage

- Let the urine drain in between IBP measurements
- Urine sampling from the needle-free port is facilitated by temporarily opening the red clamp. Remember to close clamp afterwards.
- Avoid a U-bend of the large urimeter drainage tube (which will impede urine drainage).
- Replace the FoleyManometer whenever the Foley catheter or the urine collection device is replaced, or at least every 7 days.



PANEL C, Intravesical pressure Monitoring:

- Place the "0 mmHg" mark of the manometer tube at the midaxillary line at the level of the iliac crest (mark for future reference) and elevate the filter vertically above the patient.
- Open the bio-filter clamp, and read IBP (end-expiration value) when the meniscus has stabilized after about 10 seconds.
- Close clamp after IBP measurement and place the FoleyManometer in its drainage position.

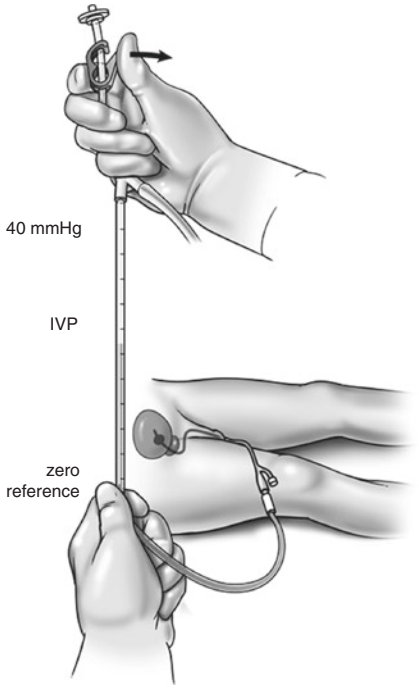


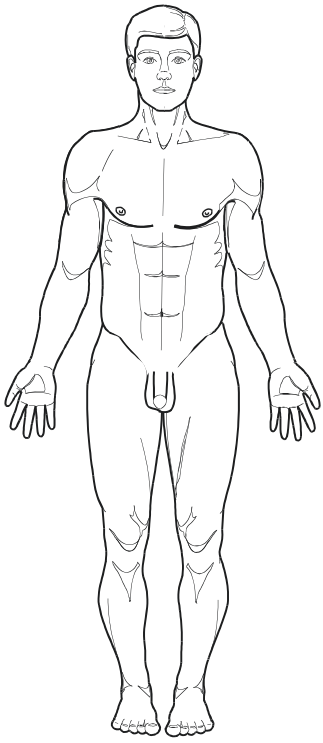
Fig. 3.2 (continued)

CENTRAL NERVOUS SYSTEM
Intracranial pressure ↑
Cerebral perfusion pressure↓
Idiopathic intracranial hypertension in morbid obesity

CARDIOVASCULAR SYSTEM[1]
Difficult preload assessment
Pulmonary artery occlusion pressure
PAOP ↑
Central venous pressure ↑
Transmural filling pressure =↘
Intra thoracic blood volume index =↘
Global end-diastolic blood volume index =↘
Extra vascular lung water =↗
Stroke volume variation ↗
Pulse pressure variation ↗
Right ventricular end-diastolic volume =↘
Cardiac output ↓
Venous return ↓
Systemic vascular resistance ↑
Venous thrombosis ↑
Pulmonary embolism ↑
Heart rate ↗ =
Mean arterial pressure ↗ =↘
Pulmonary artery pressure ↑
Left ventricular compliance ↓
Left ventricle regional wall motion ↓ ↓

HEPATIC SYSTEM
Hepatic arterial flow ↓
Portal venous blood flow ↓
Porto-collateral flow ↑
Lactate clearance ↓
Glucose metabolism ↓
Mitochondrial function ↓
Cytochrome p450 function ↓
Plasma disappearance rate Indocyanine green ↓

GASTRO-INTESTINAL SYSTEM
Abdominal perfusion pressure ↓
Celiac blood flow ↓
Superior mesenteric artery blood flow ↓
Blood flow to abdominal organs ↓
Mucosal blood flow ↓
Mesenteric vein compression ↑
Intramucosal pH ↓
Regional CO2 ↑
CO2-gap ↑
Success enteral feeding ↓
Intestinal permeability ↑
Bacterial translocation ↑
Multiple organ failure ↑
Intestinal ulcer (re)bleeding ↑
Variceal wall stress ↑
Variceal (re)bleeding ↑
Peritoneal adhesions ↑



RESPIRATORY SYSTEM
Intrathoracic pressure ↑
Pleural pressure ↑
Functional residual capacity ↓
All lung volumes ↓
(~restrictive disease)
Auto-PEEP ↑
Peak airway pressure ↑
Plateau airway pressure ↑
Dynamic compliance ↓
Static respiratory system compliance ↓
Static chest wall compliance ↓
Static lung compliance =
Hypercarbia ↑
PaO2 ↓ and PaO2/FiO2 ↓
Dead-space ventilation ↑
Intrapulmonary shunt ↑
Lower inflection point ↓
Upper inflection point ↑
Extra vascular lung water =↗
Prolonged ventilation
Difficult weaning
Activated lung neutrophils ↑
Pulmonary inflammatory infiltration ↑ ↑
Alveolar edema ↑
Compression atelectasis ↑

RENAL SYSTEM
Renal perfusion pressure ↓
Filtration gradient ↓
Renal blood flow ↓
Diuresis ↓
Tubular dysfunction ↑
Glomerular filtration rate ↓
Renal vascular resistance ↑
Renal vein compression ↑
Ureteral Compression ↑
Anti-diuretic hormone ↑
Adrenal blood flow =
Abdominal wall complications in CAPD↑

ABDOMINAL WALL
Compliance ↓
Rectus sheath blood flow ↓
Wound complications ↑
Incisional hernia ↑

ENDOCRINE SYSTEM
Release pro-inflammatory cytokines ↑ (IL-1b, TNF-a, IL-6)

[1] Cardiovascular effects are exacerbated in case of hypovolemia, hemorrhage, ischemia and high PEEP ventilation

Fig. 3.3 Pathophysiology of intra-abdominal hypertension

Conclusions

Although considerable progress relating to IAH and ACS has been made over the last decades, there is still a general lack of clinical awareness. With our presently improved understanding of the pathophysiology and epidemiology, future efforts should be largely focused on defining whether targeted or multifaceted interventions aimed at reducing IAP improve patient-important outcomes. The C_{ab} is one of the most neglected parameters in critically ill patients, although it plays a key role in understanding the deleterious effects of unadapted IAV on IAP and end-organ perfusion. If we can identify patients with low C_{ab}, we can anticipate and select the most appropriate medical or surgical treatment option to avoid deleterious complications like ACS.

Key Messages

1. The diagnosis of IAH/ACS relies on accurate IAP measurement. The current gold standard for IAP measurement is intermittently every 4–6 h via the bladder, using no more than 25 mL of instillation volume, in the complete supine position, using the midaxillary line as a zero reference point where it crosses the iliac crest.
2. There are many risk factors in relation to IAH, and they can be classified into those decreasing abdominal compliance, those increasing intra-abdominal or intraluminal contents, and those related to capillary leak and fluid overload.
3. IAP monitoring should be performed in all critically ill or injured patients exhibiting one or more risk factors for the development of IAH and continued until risk factors are resolved and IAP has remained normal for 24–48 h.
4. The incidence of IAH is around 21–58%, and the incidence of ACS is around 1–12% in critically ill patients, depending on the patient population.
5. The abdominal compliance is the most neglected parameter in critically ill patients although it plays a major role in understanding the deleterious effects of unadapted IAV on IAP and end-organ perfusion.
6. IAH and ACS cause organ dysfunction through direct compression of the heart, compression of both arterial and venous perfusion of the abdominal organs, and abdomino-thoracic pressure transmission. All organ systems in the human body are affected by IAH.

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4 The Open Abdomen: Balancing Pathophysiologic Benefits and Risks in the Era of Improved Resuscitation Practices

Derek J. Roberts, Jimmy Xiao, and Andrew W. Kirkpatrick

Key Points

- Physiological derangements after damage control (DC) laparotomy in trauma, general, and vascular surgery patients are largely related to the lethal triad and perfusion/systemic inflammatory disturbances induced by acute bowel injury and intra-abdominal hypertension (IAH) (i.e., the "acute intestinal distress syndrome").
- Both acute bowel injury and IAH are made worse by large-volume crystalloid fluid resuscitation.
- There are several proposed physiologic benefits of the open abdomen (OA). These include that it prevents the onset of (and/or interrupts) the lethal triad and ACS in patients who received conventional trauma resuscitation.
- The survival benefit of DC laparotomy has recently been questioned because of the introduction of improved resuscitation practices (such as DC

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resuscitation and the avoidance of large-volume crystalloid fluid resuscitation) and the risks associated with open abdominal management, including progressive abdominal visceral edema, loss of abdominal domain, massive ventral hernias, enteric leaks, and enteroatmospheric fistulae.
- As equipoise begins to dwindle regarding the effectiveness and safety of liberal use of damage control laparotomy in the modern era of improved resuscitation practices, randomized evidence is increasingly required to elucidate the situations in which the associated benefits of open abdominal management outweigh the risks.

4.1 Introduction

Classically, there are three broad categories of reasons why surgeons leave the abdominal cavity open: anatomical, physiological, and logistical [1]. The specific indications underlying these categories were recently systematically reviewed and then "appropriateness rated" in two international expert consensus studies and a larger survey of surgeons practicing in the USA, Canada, Australia, and New Zealand [2–5]. Appropriate anatomical indications identified in these studies included several abdominal injury patterns and an inability to close the abdominal fascia because of visceral edema [2–5]. Appropriate physiological reasons included the finding of hypothermia, acidosis, and/or coagulopathy or development of signs of intra-abdominal hypertension (IAH) and/or abdominal compartment syndrome (ACS) during attempted abdominal fascial closure [2–5]. Finally, appropriate logistical reasons involved a planned relaparotomy to remove packs, reassess bowel viability (e.g., superior mesenteric artery/vein injuries), or reestablish gastrointestinal (GI) continuity [2–5].

Open abdominal management has long been reported to be associated with a number of physiological benefits among critically ill trauma, general, and vascular surgery patients [6]. In critically injured and massively bleeding patients, one of the conventional grounding principles underlying damage control (DC) surgery is that abbreviating the procedure by conducting "rapid conservative operative techniques" and leaving the abdomen open may lead to improved patient outcomes [7]. Abbreviated surgery theoretically helps prevent further bloodshed and avoids the onset of (and/or interrupts) the "vicious cycle" of hypothermia, acidosis, and coagulopathy [3, 8, 9]. Largely through this mechanism, it has been postulated since the late 1980s that DC surgery may prevent death from "physiological exhaustion" [3, 8, 9]. Thus, use of the open abdomen (OA) (defined as "non-closure of the abdominal fascia") is a necessary component of trauma DC laparotomy [10]. Further, among those with or at risk of significant abdominal visceral edema and/or IAH (e.g., typically patients who have received a large volume of intravenous crystalloid fluid resuscitation), leaving the abdomen open after operation may prevent ACS [10, 11].

Decompressive laparotomy and open abdominal management are also the standard treatment for ACS [10–12]. Finally, recent evidence suggests that use of temporary abdominal closure (TAC) techniques that employ constant negative pressure to the open abdomen (active negative pressure peritoneal therapy [13]) may reduce peritoneal inflammation and the systemic inflammatory response syndrome (SIRS) induced by acute bowel injury and thereby prevent multiorgan dysfunction syndrome (MODS) [13, 14].

However, open abdominal management has also been associated with a high rate of energy loss and a substantial incidence of potentially severe complications (intra-abdominal sepsis, enteric leaks, enteroatmospheric fistula formation, and massive ventral hernias) [3, 15–17]. The management of these complications often requires a number of hospital readmissions and subsequent surgical procedures [3, 15–17]. Further, some have questioned whether the physiologic benefits of DC surgery will continue to be realized in the modern era of improved resuscitation practices where large-volume crystalloid fluid resuscitation is avoided [7, 18]. Thus, decisions regarding whether open abdominal management is indicated or not should be based on modern evidence or until such evidence becomes available and the opinions of experts and the currently practicing surgical community [2–5]. In this chapter, we review the pathophysiology of the OA, including the balance between its potential pathophysiologic benefits and risks. Specifically, we describe the pathogenesis of the lethal triad, acute bowel injury, abdominal visceral edema formation, IAH/ACS, SIRS, and MODS relevant to patients with an OA. Within the context of these adverse pathophysiologic changes, we also review the proposed physiologic benefits of the OA. We end with a description of the present knowledge regarding the pathophysiology of progressive loss of abdominal domain, massive ventral hernias, enteric leaks, enteroatmospheric fistulae formation, and accelerated energy expenditure in this patient population.

4.2 The Changing Playing Field

Discussion of the use of the OA in trauma, general, and vascular surgery needs to be considered within the context of recent changes in resuscitation that have likely altered the "playing field" concerning hemorrhage resuscitation. Historically, use of the OA was considered essential to avoid early death in patients with dramatic abdominal visceral edema who underwent delayed hemorrhage control after receiving a large volume of crystalloid fluid resuscitation [7, 19–22]. With fundamental changes in resuscitation practices that focus on avoidance of crystalloid fluids and use of DC resuscitation principles, there have been reports of a dramatic reduction in the extent of abdominal visceral edema, severe IAH, and ACS after emergent laparotomy [7, 23]. This changing IAH/ACS epidemiology has been coupled with reports of concerns that DC surgery and open abdominal management may be overused in the era of DC resuscitation [7]. Thus, principles that were once deemed dogma, such as the importance of judging whether a seriously injured patient would

receive an OA even before beginning an operation, are less relevant or even irrelevant in modern surgical practices [7, 24]. However, as experts and practicing surgeons have reported that there are likely still benefits of DCS in select clinical circumstances, its use should continue for these indications until randomized evidence becomes available [2–5].

4.3 The OA for Trauma Versus the OA for Sepsis

Intensive care unit (ICU) patients with an OA typically constitute a mix of injured and infected patients. Previous studies addressing management issues related to the OA have typically combined these cohorts. However, it is becoming increasingly apparent that injury and infection are significantly different pathophysiologic insults [25]. Those with infection may be disproportionately affected by persistent inflammation and its associated obligatory edema [25]. In support of this, in a recent study by Loftus et al., failure to achieve primary fascial closure (PFC) after trauma was associated with persistence of the lethal triad beyond 48 h post-injury, whereas failure to achieve PFC after infection was most related to the operative course within 48 h of the index laparotomy [25].

4.4 Pathogenesis of the Lethal Triad, Abdominal Visceral Edema, IAH/ACS, SIRS, and MODS

4.4.1 The Lethal Triad

As the original use of the OA was largely driven by the need to address the significant fatality associated with physiological exhaustion [7], any discussion of the topic requires an understanding of the lethal triad. Exsanguination, or a blood loss exceeding 40% of total body blood volume with ongoing bleeding, is often associated with development of a lethal triad (or "vicious cycle") of hypothermia (core body temperature < 34 °C), acidemia (pH < 7.2), and clinical (absence of visible clots during operation) or laboratory [e.g., international normalized ratio and/or partial thromboplastin time > 1.5 times normal] coagulopathy [2, 3, 7, 26–28]. Importantly, all three components of the lethal triad may be precipitated or exacerbated by the administration of large volumes of crystalloid fluids.

Historically, hypothermia occurred in 57–66% of severely injured patients treated with standard, crystalloid-based resuscitation practices [29, 30]. This complication typically occurred after profound shock had been resuscitated with infusion of unheated crystalloids and blood products [31]. Its occurrence was associated with a 4–41 times higher risk of intraoperative mortality [26, 29, 30, 32]. The incidence of hypothermia increases with higher injury severity scores and worsening degrees of shock. The association between hypothermia and mortality has been

reported to be stronger among those with core temperatures <33°C or who were difficult to rewarm than those who remained warm or could be timely rewarmed [29, 32]. Although it would be predicted that the incidence of hypothermia would decline as resuscitation practices abandon infusion of large volumes of crystalloid fluids, even more recent series have reported development of severe hypothermia in injured patients [33].

Metabolic acidosis often coexists with hypothermia and coagulopathy and results predominantly from anaerobic metabolism and production of lactate secondary to inadequate peripheral tissue perfusion [26, 34]. Coagulopathy was originally thought to be caused by hypothermia, metabolic acidosis, administration of a large volume of crystalloid fluids, and other factors [26, 35, 36]. However, in 2003, Brohi et al. reported that 33% of 1008 severely injured patients presenting to hospital already had prolonged clotting times that were independent of the amount of crystalloid fluids administered [7, 37]. This "acute traumatic coagulopathy" (also sometimes referred to as the "acute coagulopathy of trauma") appears to occur "before significant fluid administration, that may be attributable to the injury itself" [37]. As hypothermia and metabolic acidosis worsen coagulopathy, and ongoing bleeding secondary to coagulopathy worsens hypothermia and metabolic acidosis, the initiation of the vicious cycle often leads to progressive, diffuse, difficult to control hemorrhage and ultimately death if actions are not taken to prevent or interrupt it [3, 8, 9].

4.4.2 Abdominal Visceral Edema, IAH/ACS, SIRS, and MODS

Surgeons have long hypothesized that inflammation and abdominal visceral edema were linked, especially among patients receiving traditional (crystalloid-based) resuscitation [7]. In trauma patients receiving large-volume fluid resuscitation, the abdominal viscera can sequester liters of crystalloid fluid and has been reported to grow to more than twice the volume of the abdominal cavity after emergent trauma laparotomy [38–40]. With aggressive crystalloid resuscitation, IAH/ACS may be induced early, especially if hemorrhage is not expeditiously controlled and if hyperresuscitative strategies are used [19–21]. Thus, it is possible that many (if not most) cases of early ACS were closely linked to crystalloid-focused resuscitation strategies in the past [41, 42]. However, despite the decline in use of crystalloid fluids and associated increase in use of blood product in recent years, overt cases of ACS continue to be reported.

Abdominal visceral edema and IAH/ACS may result from a two "hit" pathophysiologic process named the "acute intestinal distress syndrome" in 2008 by Malbrain and De Laet [11, 43–45]. In the first "hit," resuscitation of patients with hypovolemic or septic shock produces an ischemia–reperfusion injury of the bowel [11, 38, 39, 46]. This "acute bowel injury" results in upregulated transcription, translation, and release of a number of pro-inflammatory mediators [e.g., tumor necrosis factor-alpha, interleukin (IL)-1, and IL-6) into the

peritoneal cavity [11, 38, 44]. In patients with peritonitis, peritoneal inflammatory mediator concentrations have been reported to be 10–1000 times higher than levels in blood [47].

These mediators promote neutrophil priming and increase intestinal wall and mesenteric capillary permeability, which results in translocation of bacteria across the intestinal wall, release of bacterial endotoxin, and extravasation of fluid into the bowel wall, its supporting mesenteries, and the peritoneal cavity [11, 38, 39, 48–54]. Further, the pro-inflammatory mediators produced during the acute intestinal distress syndrome may also adversely affect distant organs [47]. Mediators absorbed across the bowel wall/peritoneum, via intra-abdominal portal or systemic veins, or mesenteric lymphatics in the bowel wall, the cisterna chyli, and the thoracic duct have been reported to induce SIRS and MODS. As the gut represents the largest surface area in contact with the external environment (and constitutes a reservoir of over 100 trillion bacteria), this process may serve as the "motor" that drives MODS during critical illness [55].

In the second "hit" of the acute intestinal distress syndrome, abdominal visceral edema (and intra-abdominal packing, hematomata, and/or ascites, if present), along with a likely concurrent reduction in abdominal wall compliance among the critically ill/injured, produces IAH [11, 38, 39, 56, 57]. IAH compresses intra-abdominal lymphatics, decreasing lymph flow out of the abdominal cavity [38, 39]. It also decreases bowel wall arterial inflow and venous outflow, resulting in a progressive (mucosa-to-serosa) intestinal wall necrosis and a further increase in bowel wall permeability, elevated bacterial translocation/systemic endotoxin absorption, and heightened release of pro-inflammatory mediators [11, 38, 39, 58]. This results in a further increase in intestinal wall permeability, abdominal visceral edema, and intra-abdominal pressure (IAP), especially when patients continue to receive massive crystalloid fluid resuscitation [11, 38]. In doing so, these events initiate a self-perpetuating process of progressive acute bowel injury, abdominal visceral edema, and IAH (the acute intestinal distress syndrome), ultimately culminating in the ACS [11, 38].

Ironically, the effects of IAH on the gut are similar to those of prolonged hypoperfusion, and, therefore, these two issues are compounding. Even after resuscitation and normalization of hemodynamics, gut vasoconstriction persists and is further exacerbated by IAH. Prolonged gut hypoperfusion can precipitate a severe inflammatory response due to mobilization of damage-associated molecular patterns (e.g., high mobility group box 1, heat shock proteins, s100 proteins, nucleic acids, and hyaluronan), pro-inflammatory cytokines, and other mediators [59]. Thus, the OA may have a profound effect on reducing the progression of severe injury/infection with shock to MODS through ameliorating injury to the gut. However, it remains unknown if the benefit of the OA in this setting results from correcting IAH or draining inflammatory ascites. There is a complex relationship between pressure, ischemia, and inflammation within the peritoneal cavity. Independently, the damaged gut seems to act as a continued source of inflammation propagating SIRS and potentiating MODS [60–62].

4.5 Proposed Pathophysiologic Benefits of the OA

Use of the OA allows laparotomy to be quickly terminated, which in theory limits surgical time, tissue injury, blood and temperature loss, and fluid and blood product requirements. Although this is widely believed to assist in avoiding and/or interrupting the lethal triad in patients with the most severe injuries or signs of impending physiological exhaustion, limited studies exist to support this. Further, as many severely injured patients likely present with acute traumatic coagulopathy, DC surgery is likely incapable of preventing coagulopathy in the majority of the critically injured [7]. Finally, as the development of the lethal triad in earlier studies of DC surgery and open abdominal management were related to the use of standard, crystalloid-based resuscitation, and the lethal triad may be prevented with the use of hemostatic resuscitation strategies, studies are urgently required to determine the role of DC surgery and open abdominal management in the context of changing resuscitation practices [7].

A now long-proposed benefit of the use of the OA is that it prevents against development of the ACS after DC laparotomy [7, 63]. However, as outlined by Balogh and colleagues, cases of overt ACS requiring decompressive laparotomy are becoming increasingly rare in many practice settings as this syndrome has been largely "eliminated" by "strategic research and focused preventions," especially the avoidance of over-resuscitation with crystalloid fluids [64]. Thus, this indication may become increasingly less important in modern day surgical practice and the current era of improved fluid resuscitation strategies. When needed, decompressive laparotomy has been reported to result in nearly immediate and sometimes substantial improvements in organ dysfunction in patients with the ACS. Despite this, careful (but retrospective) study has revealed that although IAP is consistently lower after decompression, mortality remains considerable, and recuperation of organ dysfunction after decompressive laparotomy for ACS is variable [65].

Finally, although limited clinical evidence yet exists, studies have suggested that active negative pressure peritoneal therapy TAC techniques may remove peritoneal cytokines, reduce systemic inflammation, and improve outcomes in trauma and emergency general surgery patients with an OA. These types of TACs include noncommercial (e.g., the Barker's vacuum pack) and commercial devices. A study allocated animals with a fecal blood clot model of intra-abdominal sepsis to negative pressure peritoneal therapy [the Kinetic Concepts Inc. (KCI) vacuum-assisted closure device, KCI, San Antonio, TX, U.S.A.] versus passive drainage of the peritoneal cavity and observed reduced levels of systemic pro-inflammatory cytokines and improved intestine, lung, kidney, and liver histopathology [14, 52]. A multicenter prospective cohort study reported in 2013 that use of the ABThera Open Abdomen Negative Pressure Therapy Device (KCI) after abbreviated laparotomy in patients with intra-abdominal injury or sepsis was associated with improved adjusted survival among when compared to the Barker's vacuum pack (Fig. 4.1) [14, 66]. This concept was subsequently further examined by a single-center randomized controlled trial (RCT) that allocated patients who underwent DC laparotomy for abdominal trauma or intra-abdominal sepsis to the ABThera versus Barker's vacuum pack technique [13, 14]. Interestingly, this study observed no significant

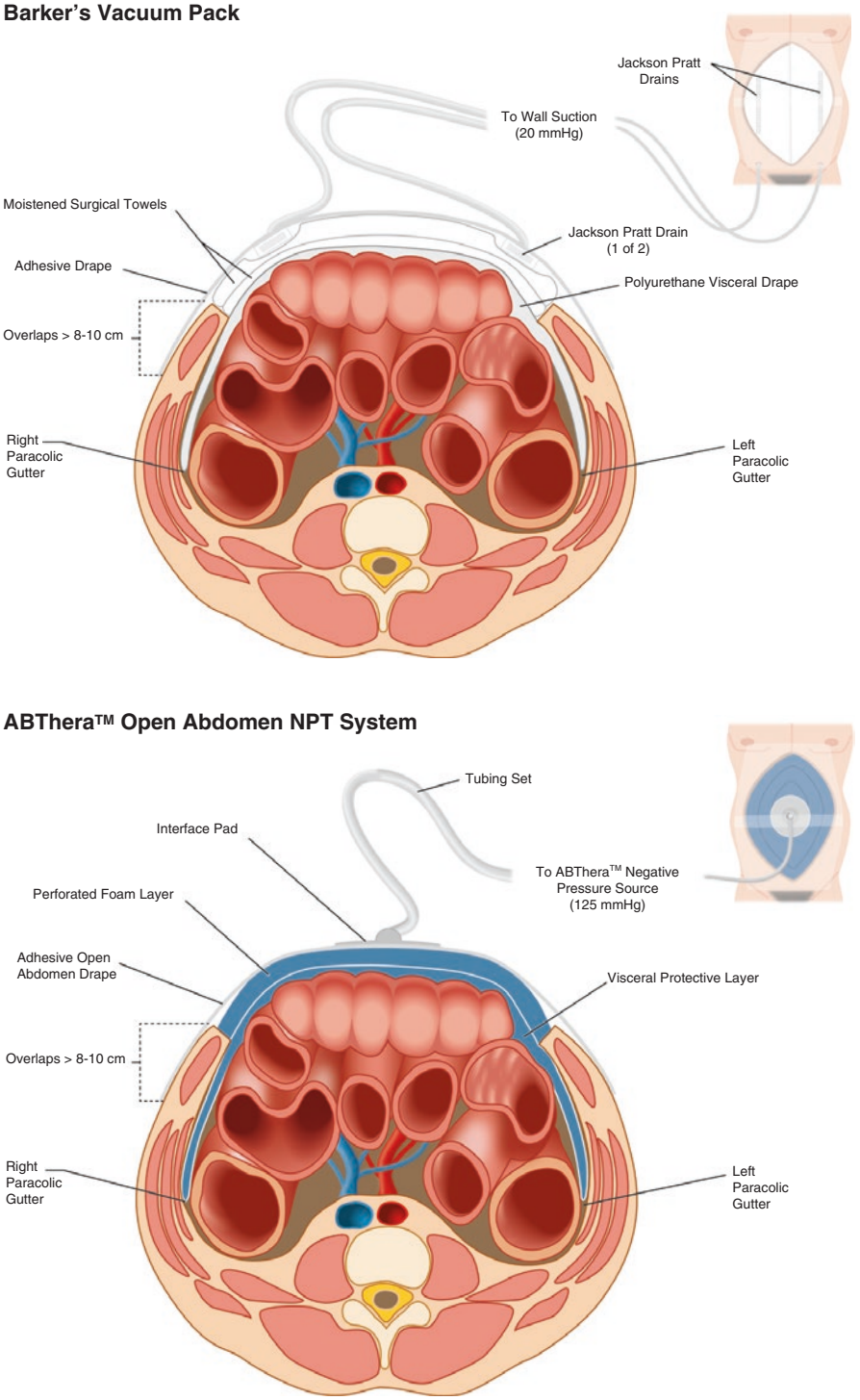

Fig. 4.1 Schematic of the Barker's vacuum pack (*left*) and ABThera Open Abdomen Negative Pressure Therapy System (*right*). Illustration reproduced from reference [13]

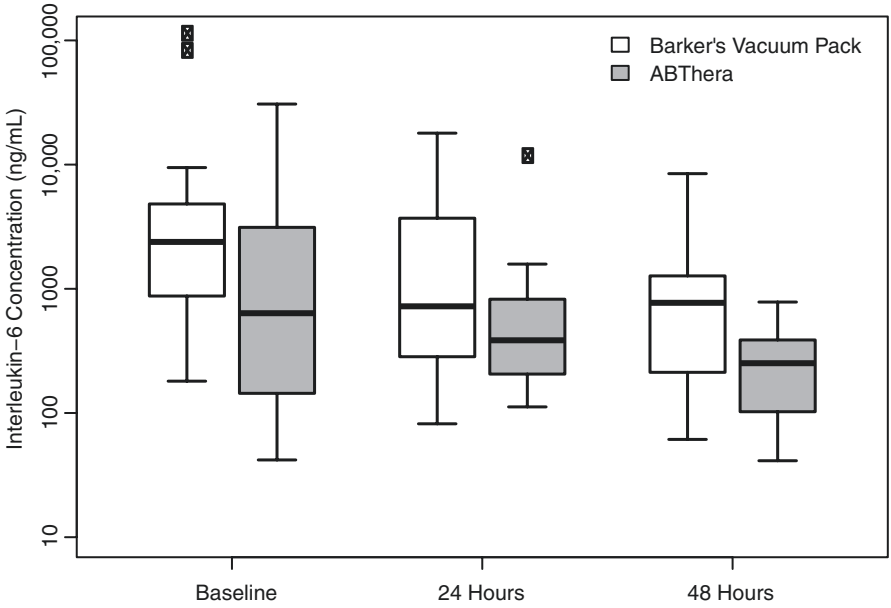

Fig. 4.2 Plasma interleukin-6 (IL-6) levels in a randomized controlled trial that allocated adults who underwent abbreviated laparotomy for intra-abdominal injury or sepsis to the ABThera Open Abdomen Negative Pressure Therapy System or the Barker's vacuum pack. Figures reproduced from reference [14]

difference in the plasma concentration of IL-6 and five other pro-inflammatory cytokines at baseline versus 24- or 48-h between groups (Fig. 4.2) [14]. However, the intention to treat analysis revealed a significantly improved 90-day survival in the ABThera versus Barker's vacuum pack group (hazard ratio = 0.32 for mortality) (Fig. 4.3) [14]. As the findings of improved survival could be the result of residual confounding or type I error, the authors suggested that further work was required to explain the potential mechanisms of improved outcomes and confirm the findings before they are used to inform surgical practice [14].

4.6 Pathophysiology of the OA and Its Associated Complications

There are several potential complications of open abdominal management. The proposed pathophysiology of some of the most significant of these complications is discussed below.

4.6.1 Progressive Abdominal Visceral Edema, Loss of Domain, and Massive Ventral Hernias

Patients who have suffered an acute bowel injury are likely particularly susceptible to fluid shifts during the early postoperative period after the index laparotomy for

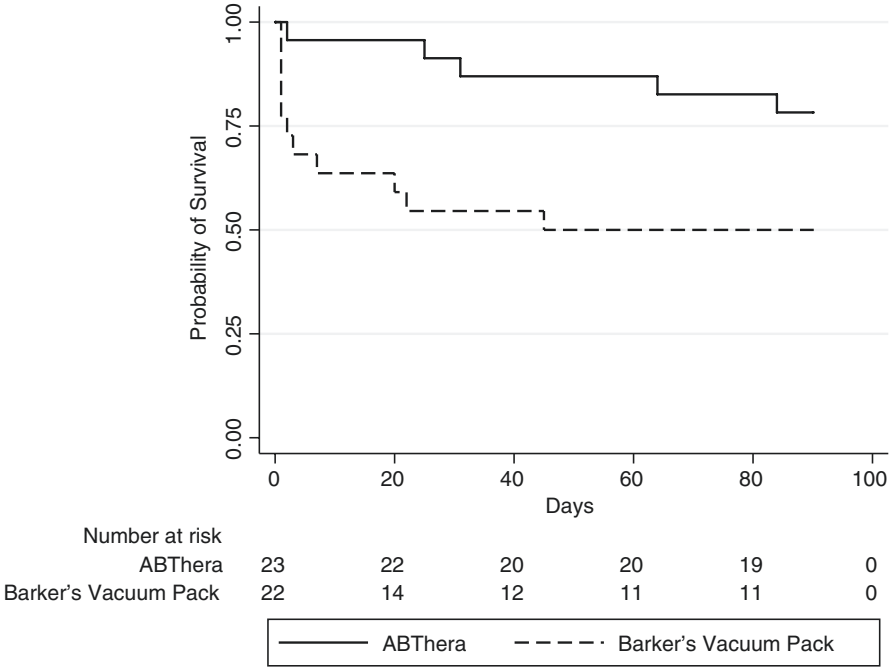

Fig. 4.3 Survival probability in a randomized controlled trial that allocated adults who underwent abbreviated laparotomy for intra-abdominal injury or sepsis to the ABThera Open Abdomen Negative Pressure Therapy System or the Barker's vacuum pack. Figures reproduced from reference [14]

open abdominal management [67]. Historically, they were also likely to receive a high volume and/or rapid infusion of crystalloid fluids during this period [67]. As fluid follows the path of least resistance, having an open abdominal cavity during this time may decrease resistance to flow and likely facilitates a rise in abdominal visceral edema and distention of abdominal contents that might not otherwise have occurred in a closed abdominal cavity [67]. This may result in a progressive increase in abdominal visceral edema (and an associated progressive decrease in the probability of primary fascial closure) after the index laparotomy [67]. In support of this argument, studies have suggested that if the abdomen is not closed early, the incidence of PFC declines significantly. In a prospective, multicenter cohort study, Pommerening et al. reported that after postoperative hour 24, each subsequent hour delay in returning to the operating room was independently associated with a 1% decrease in the odds of PFC [67].

If the abdomen is not closed within 10–14 days, a dense network of inflammatory and granulation tissue develops between bowel loops, the greater omentum, and the parietal peritoneum on the undersurface of the abdominal wall [7, 68]. This tissue forms a firmly adherent superficial layer over the entire viscera and acts to progressively obliterate the intraperitoneal space, creating a "frozen abdomen" [7, 68].

This process, when combined with the gradual, obligatory lateral retraction of the abdominal fascial edges and shortening of the abdominal wall muscles (which occurs because the lateral forces evoked by the oblique and transverse muscles overcome the medial forces evoked by the ipsilateral rectus muscles once the linea alba has been divided), both contribute to a progressive loss of intra-abdominal domain [7]. In the absence of surgical or TAC techniques that apply constant medial tension to the midline abdominal fascia, abdominal visceral edema and progressive lateral fascial retraction result in loss of domain and ultimately a massive ventral hernia.

4.6.2 Enteroatmospheric Fistulae and Enteric Leaks

With increased swelling, manipulation, trauma, desiccation, and infection of the bowel, enteroatmospheric fistulae or enteric leaks may form either superficial or deep within the abdominal cavity [7]. As outlined by Björck and colleagues, an enteroatmospheric fistula represents an enteric fistula within the middle of the open abdomen (with defining characteristics including the absence of a fistula tract, the lack of well-vascularized surrounding tissue, and spillage of enteric content directly into the peritoneal cavity) [10, 69]. In contrast, an enteric leak is characterized by spillage of enteric contents into the abdomen without established enteric fistula development [10, 69].

The pathophysiology underlying the formation of enteroatmospheric fistulae is inadequately understood. In a retrospective cohort study of 517 trauma patients with an open abdomen after DC laparotomy conducted across 14 trauma centers in the USA, Bradley et al. reported that independent predictors of the development of enteroatmospheric/enterocutaneous fistulae or intra-abdominal sepsis included large bowel resection, a total fluid intake at 48 h >5 L, and an increasing number of abdominal re-explorations [70]. Thus, crystalloid fluids, bowel manipulation, and local visceral trauma during repeated laparotomies may contribute to development of edematous and friable bowel and increase risk of enteroatmospheric fistulae and enteric leaks in patients with an OA [70–72]. Finally, although it remains unknown if this relationship exists in humans, the length of peritoneal air exposure has also been reported to be associated with a proportional increase in damage of the gastrointestinal tract in rats [73].

4.6.3 Energy Expenditure

The OA is associated with increased fluid and protein loss, which can produce nutritional insufficiency and a catabolic state [74]. A prospective cohort study by Cheatham et al. reported that nutritional calculations that fail to account for protein/nitrogen loss may underestimate actual nitrogen balance by an average of 3.5 g/24 h [75]. These authors suggested that in the absence of actual direct measurement of losses, an estimate of 2 g of nitrogen per liter of abdominal fluid should be included

in any nutritional calculations. However, with advanced TAC dressings, this concern may be less important. A recent RCT utilizing active negative pressure peritoneal therapy reported less fluid drainage compared to a less efficient TAC, with hypotheses being suggested that NPPT might actually reduce inflammation and thus edema generation [14].

As early enteral nutrition (EN) improves wound healing, decreases catabolism, preserves gastrointestinal integrity, and reduces septic complications and lengths of stay [75, 76], patients with an OA should be fed early. Despite this, there has been a reluctance to start feeds in this population, likely due to perceived concerns regarding the potential to exacerbate a preexisting ileus, visceral swelling, or clinical inattention to the benefits of EN. However, administering EN has been shown to be feasible in patients with an OA [74] and may even increase visceral blood flow [77], which has been speculated to mitigate bowel edema and facilitate PFC. Further, anything that preserves the integrity of the gut mucosal barrier and modulates inflammatory mediator generation presumably mitigates bowel swelling and therefore acts to improve the rate of PFC.

Conclusion

Surgeons leave the abdomen open for anatomical, physiological, and logistical reasons. Physiological derangements after damage control laparotomy are largely related to perfusion/systemic inflammatory disturbances induced by acute bowel injury and IAH. Importantly, both of these derangements are made worse by large-volume crystalloid fluid resuscitation. Potential physiologic benefits of the OA have long been hypothesized to include prevention or improvement of the adverse effects of ACS. The OA also prevents the more subtle consequences of IAH, facilitates use of active negative pressure peritoneal therapy, allows for delayed reconstructive options when abdominal domain has been lost, and permits planned abdominal re-exploration to remove sponges and reestablish intestinal continuity. However, the survival benefit of DC laparotomy has recently been questioned because of the introduction of improved resuscitation practices (such as DC resuscitation and the avoidance of large-volume crystalloid fluid resuscitation) and the risks associated with open abdominal management, including massive ventral hernias, enteric leaks, and enteroatmospheric fistulae. As equipoise begins to dwindle regarding the effectiveness and safety of liberal use of damage control laparotomy in the modern era of improved resuscitation practices, randomized evidence is increasingly required to elucidate the situations in which the associated benefits of open abdominal management outweigh the risks.

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Indications for Open Abdomen in the Non-trauma Setting

5

Hany Bahouth and Yoram Kluger

5.1 Introduction

The principles of damage control laparotomy (DCL) in the trauma setting are well described. Although the indications for this technique vary among care providers, it is agreed that that this approach revolutionized the management of critically injured patients [1, 2]. Overall, about 10–15% of all laparotomies for trauma are managed with damage control techniques [3].

It has been suggested that DCL principles could apply to acute surgical non-trauma conditions. In non-trauma setting, the causes for the physiologic derangements observed are diverse and different from that of the injured. Despite this deference, the basic pathophysiology that results in acidosis, coagulopathy, and hypothermia remains the same.

With better understanding of damage control principles, the open abdomen strategy, first described almost 120 years ago by McCosh [4], became a common and appreciated philosophy practiced enthusiastically by surgeons in non-trauma emergency general surgery [5].

In spite of the adoption of the open abdomen technique in non-trauma emergency surgery, the indications that define its appropriate application are poorly agreed among surgeons [6–11]. This is due to the lack of well-constructed randomized studies exploring the concept in the non-trauma settings as well as deficiency of common language and classification system of surgical emergencies that may favor from open abdomen strategies. Additionally, diverse practice patterns

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described in various published studies and the inclusions of heterogeneous patient population contribute to the paucity of evidence-based data [6–8, 11]. Several challenging clinical and surgical circumstances may indeed benefit from the use of DCS and the open abdomen philosophy in the non-trauma setting.

In their review, looking at the indications for open abdomen in non-trauma surgery, Atema et al. [12] included 78 series with overall 4358 patients. Secondary peritonitis was diagnosed in 3461 (79%). The most frequent single indication for the open abdomen management was a planned relaparotomy strategy.

There are no definitive physiological or anatomical criteria to aid surgeons to properly consider and select damage control strategy in non-trauma surgical patients. Nevertheless some of the indications and physiological-based criteria were extrapolated from the trauma arena; temperature lower than 35°C, pH less than 7.20, or base deficit exceeding eight and laboratory or clinical evidence of coagulopathy, need for vasopressors, and massive blood transfusions. These values represent patients failing to compensate for shock state.

Neto et al. [13] proposed three basic considerations for the application of the open abdomen strategy:

(a) **Anatomical** (soft tissue loss, impending risk of abdominal compartment syndrome)
(b) **Physiological** (pertain to a severe physiologic derangement)
(c) **Logistical** (necessity for serial surgical interventions)

Success rates of delayed fascial closure are lower in non-trauma patients compared to trauma patients. Several studies identified peritonitis as an independent predictor of failure of fascial closure [14–16].

Higher fascial closure rates were achieved at the first re-exploration and less likely during the second or third take back [17].

Shorter period of open abdomen was also associated with higher fascial closure rates [18–20]. Moreover, a preemptive open abdomen for intra-abdominal hypertension or for decompression of established ACS is associated with higher fascial closure rates [21]. The mortality rates of patients submitted for the open abdomen exceed 30% and are dependent on the causative event leading to the selection of the open abdomen strategy [22].

In this chapter, we will review the most common indications for the open abdomen technique in the non-trauma setting and their utility and efficacy.

5.1.1 Open Abdomen for Intraperitoneal Sepsis\Peritonitis

The role of the open abdomen in the management of severe secondary peritonitis is controversial. Based on small retrospective studies in the 1980s and 1990s, enthusiastic adoption of the strategy for patients diagnosed with severe peritonitis was recorded [23, 24].

Physiologic instability, temporary source control, and avoidance of abdominal compartment syndrome were the cornerstone indications for damage control in the septic abdomen. Most abdominal sepsis results from pathologies in the hepatobiliary tree, pancreatic necrosis/infection, or hollow viscus perforation/necrosis. The main surgical focus in the septic abdomen should be therefore to obtain source control by means of peritoneal lavage, debridement of infected/necrotic tissue, and definitive or temporary measures to control anatomic integrity and to restore optimal function [25].

In severe secondary peritonitis, a staged approach may be compulsory for three different reasons or any of their combination [26].

First, the inability to control the source of contamination in a single operation; second, if the surgeon feels the patient will not tolerate a definitive repair and/or abdominal wall closure due to the severe physiological derangement [27]; and third, the presence of extensive visceral edema that may increase the risk of ACS development, if primary fascial closure is attempted [28].

Aggressive resuscitation needed in the septic patient in preparation for induction of anesthesia has led to an increased incidence in abdominal compartment syndrome. The use of the open abdomen may therefore allow potential prevention of this complication [29–32]. Two strategies in the management of these difficult patients have been reported [33, 34]: relaparotomy on demand (when required by the patient's clinical condition) and planned relaparotomy, within 24–48 h after the index operation. Subsequent studies failed to show any significant benefit of one of these strategies over the other. In a prospective, open, nonrandomized trial, sponsored by the Surgical Infection Society, 239 patients with surgical abdominal infection were treated with either the open abdomen technique or laparotomy on demand. No significant difference was observed in mortality between the two groups [35]. In 2007, Robledo and colleagues [36] randomized 40 patients with severe secondary peritonitis to open abdomen group and a laparotomy on-demand group. The mortality differences did not reach statistical significance. The authors concluded that closed management of the abdomen could be a more rational approach in the management of severe peritonitis.

Van Ruler et al. [37] in a randomized controlled trial looked at patients with secondary peritonitis and compared the differences between on-demand versus planned relaparotomy. They found no significant difference in mortality and morbidity between the two groups. However, there was a significant reduction in the number of procedures, shorter hospital stay, and decreased cost of health care in the on-demand group. It is important to note that in this study, patients who underwent abdominal packing or stapled off ends of bowel were excluded. It could be argued that these are the main target patient group for DCL in the first instance.

However, recent experimental and clinical work has suggested that the open abdomen technique with temporary abdominal wall closure using negative pressure therapy (NPT) methods is associated with superior outcomes. Amin et al. [38], in a prospective analysis of 20 patients treated with NPT following laparotomy for severe peritonitis, reported 100% survival. The study concluded that NPT is safe, but further research is needed.

Horwood and colleagues [39], in a study of 27 patients who were treated with open abdomen and NPT, reported significantly improved observed survival compared with expected survival based on P-POSSUM (physiological and operative severity score for the enumeration of mortality and morbidity) evaluation. The study concluded that open abdomen approach with immediate NPT is effective in managing patients with severe peritonitis.

Kubiak and colleagues [40], in an experimental porcine model with intestinal ischemia/reperfusion and peritoneal fecal contamination, showed that the open abdomen combined with NPT reduced mortality and organ dysfunction compared with animals treated with the traditional passive drainage.

Brandon et al. reported 338 primary laparotomies that were performed during the 1-year study period, of which 96 patients (28%) were managed with open abdomen [41]. In-hospital mortality for the open abdomen patient population was 30%. The most common preoperative indications in the index laparotomy leading to selection of the open abdomen strategy were perforated viscus and/or the presence of extra luminal gas on abdominal imaging (20%), mesenteric ischemia (17%), peritonitis and/or septic abdomen (16%), and gastrointestinal hemorrhage (12%). Damage control surgery (n = 35, 37%) was the most commonly reported indication for the decision to proceed with an open abdomen.

In summary, there is reasonable clinical evidence that planned relaparotomy does not have a survival benefit compared to on-demand laparotomy based on several studies, including one large meta-analysis. The open abdomen in abdominal sepsis might be associated with increased mortality and a higher incidence of enteroatmospheric fistulae compared to relaparotomy on demand [37, 42–48]. On-demand laparotomy is associated with decreased costs and health-care use [47, 48].

Unfortunately, the studies dealing with this controversy demonstrate substantial variability, and therefore their conclusions are scrutinized with caution.

Hemodynamics and the need for vasopressors support of the patients submitted to emergency surgery due to abdominal sepsis should guide the acute care surgeon to consider damage control and open abdomen techniques.

5.1.2 Abdominal Compartment Syndrome

Intra-abdominal hypertension (IAH) has been recognized as a clinical entity for nearly 150 years [49, 50]. The term “abdominal compartment syndrome” (ACS) was coined by Fietsam in 1989.

In 2004, the World Society of the Abdominal Compartment Syndrome (WSACS) [50] published the guidelines for the definition, diagnosis, and management of IAH and ACS.

5.2 Definitions

5.2.1 Intra-abdominal Pressure

IAP is the steady-state pressure within the abdominal cavity [50]. In healthy individuals, IAP range is 0–5 mm Hg and varies inversely with intrathoracic pressure during normal breathing.

5.2.2 Intra-abdominal Hypertension

IAH is a sustained or repeated pathological elevation of IAP of 12 mm Hg or greater [50].

WCACS introduced a useful grading system for IAH: Grade I (IAP 12–15 mm Hg), Grade II (IAP 16–20 mm Hg), Grade III (IAP 21–25 mm Hg), and Grade IV (IAP > 25 mm Hg). In contrast with IAH, ACS is not graded.

5.2.3 Abdominal Perfusion Pressure

Abdominal perfusion pressure (APP) is a measure of the relative adequacy of abdominal blood flow. APP is calculated by subtracting the IAP from the mean arterial pressure (MAP): MAP-IAP = APP. The APP in patients with IAH or ACS should be maintained at 60 mm Hg or higher [50–52].

5.2.4 Abdominal Compartment Syndrome

ACS is a sustained IAP greater than 20 mm Hg (with or without an APP < 60 mm Hg) associated with new organ dysfunction or failure [50–52].

5.2.5 Etiology of ACS

WSACS categorizes conditions that cause ACS as primary (surgical), secondary (medical), and recurrent [52].

Primary ACS is most commonly encountered and occurs from a primary intra-abdominal pathology.

Secondary ACS refers to ACS that results from massive bowel edema from sepsis, capillary leak, massive fluid resuscitation, or burns. Recurrent ACS occurs after resolution of primary or secondary ACS.

5.2.6 Incidence and Prevalence

Malbrain et al. [53] conducted a prevalence study in 13 intensive care units (ICUs) and assessed 97 patients. The overall prevalence of IAH was 58.8%. Prevalence was 65% in surgical patients and 54.4% in medical patients.

Vidal et al. (14:6) studied the incidence of IAP in 83 critically ill patients in a single ICU. A total of 31% of the patients had IAH at the time of admission to the unit, and the condition developed in another 33% after admission. ACS developed in ten patients (12%), and eight of the ten (80%) died. Brandon et al. had a total of 338 primary laparotomies that were performed during the 1-year study period, of which 96 patients (28%) were managed with an OA/ 6% due to ACS [8].

Reintam et al. [54] enrolled 257 patients receiving mechanical ventilation to identify the differences in incidence, course, and outcome of primary and secondary IAH and to determine if IAH is an independent risk factor for death. ICU mortality was 37.9% for patients with IAH and 19.1% for patients without IAH ($P = 0.001$). The authors [55] concluded that development of IAH is an independent risk factor for death.

5.2.7 Management and Treatment

Sedation, analgesia, and neuromuscular blockade can be used to decrease IAP. Drainage of any significant ascites may reduce the IAP.

Abdominal decompression via decompressive laparotomy is the standard treatment of refractory IAH and ACS. A transverse bilateral subcostal incision may be elected or the subcutaneous linea alba fasciotomy (SLAF) technique, described by Leppäniemi et al. [56].

The goals of decompressive laparotomy (DL) include decrease of increased IAP to stop organ dysfunction, to allow the continued expansion of abdominal viscera and to provide temporary abdominal coverage. With decompression, there is an immediate decrease in IAP; however, the pressure may not decrease to normal levels. An immediate recovery for some organs can be noticed. Despite the positive hemodynamic consequences of DL, a 6-year study from England showed that there was no significant difference in pre- and postoperative organ dysfunction score after DL [57].

However, Raeburn et al. found that the development of ACS in cases in which the fascia was closed, the skin only was approximated, or a Bogota bag was used was equivalent [58].

5.3 Summary

ACS is a life-threatening condition. High index of suspicion for proper diagnosis before the onset of organ failure and prompt treatment once the diagnosis is made will result in favorable outcome. The classic syndrome of cardiopulmonary and

splanchnic abnormalities represents advanced stage, and surgical decompression should not be delayed until this stage is reached [59–62].

5.3.1 Acute Severe Pancreatitis

Sever acute pancreatitis (SAP) is defined by the presence of persistent organ failure for >48 h. SAP develops in 15–20% of the patients [63–65].

The incidence of intra-abdominal hypertension (IAH) in patients with severe acute pancreatitis (SAP) is approximately 60–80%. Chen et al. [66] and Al-Bahrani et al. [67] reported an incidence of approximately 60%, using the current definitions of IAH according to the (WSACS) [68]. When the current WSACS definition was used, the reported incidence of abdominal compartment syndrome (ACS) in these patients was 27%. Dambrauskas et al. reported IAH in 58% and ACS in 19% of patients with severe acute pancreatitis [69].

There is growing evidence in the literature that development of abdominal compartment syndrome (ACS) in patients with severe acute pancreatitis (SAP) has a strong impact on the course of disease [69–72]. The mortality rate in patients developing ACS remains high, 50–75% despite several interventional options. The mortality was 11% in patients without this complication [73]. IAH in severe acute pancreatitis is reported to be an early phenomenon. A very recent prospective study from Scotland evaluating 218 patients with AP estimated that 14% had IAH on admission and another 3% developed IAH during hospitalization [74]. Up to 70% of patients have been reported to have IAH on admission to the ICU. The duration of IAH is more important than the absolute increase in IAP [52]. The etiologies for the development of IAH during SAP are several: visceral edema, ascites, acute peripancreatic fluid collection, paralytic ileus, duodenal obstruction causing gastric dilatation, and aggressive fluid resuscitation. The abdominal wall may also be edematous with decreased compliance, which in synergy with increased intra-abdominal volume leads to IAH.

5.3.2 Management of IAH & ACS in Patients with Severe Acute Pancreatitis

The management approach to LAH & ACS is nonoperative or operative [24].

Initial and immediate steps should be nonoperative. The head of the patient's bed should be raised not more than 30 degrees, neuromuscular blockage should be administrated [75], and relief of pain and anxiety with proper medication should be initiated promptly [52]. Enteral decompression with nasogastric is helpful in managing ileus and gastric dilation frequently encountered in SAP [76]. Prokinetic agents such as erythromycin and metoclopramide may help in avoiding paralytic ileus [77]. Administration of loop diuretics to enhance fluid removal may be of benefit if patients' hemodynamic allow its administration. Removal of fluid by extracorporeal techniques is more effective and may have an immediate effect [78].

It remains unclear whether this has any influence on IAH [7, 79].

Percutaneous drainage catheter (PCD) may relieve ACS in patients with intra-abdominal ascites, collections, or abscesses [80]. It is unclear if this procedure can achieve sufficient decompression and relief of the ACS.

The efficacy of the nonoperative management of ACS is not well known. In a study including 74 patients with SAP, 20 patients developed ACS during the first 7 days of admission [72]. Seven patients responded to nonoperative therapy, but the remaining 13 patients had progressive deterioration of organ dysfunction and received interventional decompressive procedure.

5.3.3 Indications for Surgical Intervention

The indications for surgical decompression in ACS associated with severe acute pancreatitis are not clear, and the IAP is not the only parameter for this decision [81–83].

It is will accepted that when nonsurgical interventions fail to relieve IAH in the presence of fulminate ACS, especially in the setting of persistent organ dysfunction, surgical decompression is justified [84].

The effects of abdominal decompression on organ functions were summarized in a collective review of 250 patients who underwent midline laparotomy [83, 85]. Decompression had a positive effect on hemodynamic, respiratory, and renal function parameters. Boone et al. [83] reported statistically significant improvements in several physiologic parameters. However, despite initial improvement almost in all patients, mortality rate of 50% was recorded. In another retrospective study, the mortality after various techniques of decompression was 46% [83].

5.3.4 Summary

IAH and ACS are common complications in acute severe pancreatitis. Early monitoring, diagnosis, and management improve outcome. Different surgical techniques exist but currently there are no randomized trials comparing the outcomes of the different surgical approaches.

5.3.5 Acute Mesenteric Ischemia

Acute mesenteric ischemia (AMI) is a challenging clinical and surgical pathology with high morbidity and mortality rates. Despite the improvements in diagnostic studies and management options, the mortality from AMI ranges between 60 and 90% [86] especially in elderly patients.

Acute arterial occlusion is the most common cause of mesenteric ischemia and results from embolic occlusion in 40–50% and thrombotic occlusion in 20–35% of the patients [87].

Due to its insidious onset and the lack of a typical clinical presentation, AMI is usually diagnosed when peritonitis becomes evident. Since bowel resection is a common treatment for AMI, to assess the final bowel viability accurately, a second-look laparotomy remains gold standard. This policy includes a routine re-exploration of the abdomen 24–48 h after the index operation carried out in an effort to preserve as much bowel as possible. At the index operation, only frank necrotic bowel is resected. However, the exact indication for second-look laparotomy is still controversial.

Some surgeons select aggressive approach with a scheduled second-look procedure in any patient who undergoes bowel resection and primary anastomosis [88, 89], whereas others suggest a more selective approach [90, 91]. Sachs et al. reported that second-look laparotomy benefits only 18–20% of patients operated for AMI. Other researchers found that fewer than half the patients underwent a second-look operation and more than 40% benefited from the procedure that resulted appropriate treatment. In mesenteric venous thrombosis, the thrombotic process extends well beyond what appears to be the compromised bowel. Therefore, a second-look exploration is often the only way to establish the full extent of nonviable bowel.

In a nonrandomized case–control study [92], patients undergoing planned relaparotomy were matched with patients who underwent relaparotomy on demand. There was no significant difference in mortality between the groups, but multiple organ failure and septic complications were more common in the patients who underwent planned relaparotomy.

Second-look laparotomy may be avoided if the experienced surgeon identifies clear margins of demarcation between well-vascularized and necrotic bowel in a hemodynamically stable patient.

5.4 Summary

The main problem in defining the role of the second-look operation for AMI is the lack of prospective studies comparing the outcomes of second-look operations with a control. Until large prospective studies are available, the indications for a second-look operation should be evaluated with caution and be based on surgeon experience and based on the surgical findings as well as patient hemodynamics.

5.4.1 Open Abdomen for Ruptured Abdominal Aortic Aneurysm

The vascular surgeon Irving Kron was the first to describe ACS following ruptured abdominal aortic aneurysm (RAAA) repair [93].

ACS develops in these patients due to aggressive resuscitation after major bleeding. Survival may be improved if the hypoperfusion of the abdominal organs resulting from IAH/ACS be reversed in time [94–96].

Routine measurement of IAP will demonstrate that IAP > 20 mm Hg occurs in 50% of all patients after open repair of a RAAA, and 20% will go on to develop

ACS [96–98]. In conclusion, IAH/ACS is a common problem after RAAA repair, in particular in the unstable patient requiring massive transfusions, irrespective of which method of repair is used.

Damage control principles [2, 96–98] in emergency surgery, as well as the modern blood resuscitation strategies [99], the development of massive transfusion protocols, and permissive hypotension management, are recent advances in the management of the bleeding vascular patient that may decrease the risk of IAH/ACS.

Mell et al. [99] showed that patients who received less than one unit of plasma for every two units of red blood cells during RAAA repair had a four times higher mortality rate than those receiving more plasma resuscitation highlighting the importance of a massive transfusion protocol.

With the introduction of endovascular aneurysm repair (EVAR) [100, 101] by Volodos in 1986, and the application of this technology on patients with ruptured abdominal aortic aneurysm (AAA), first described by Ohki and Veith [102], the management of RAAA repair has changed.

Wanhainen et al., in a multicenter prospective cohort study in four Swedish hospitals assessing the risk of requiring open abdomen treatment in patients with RAAA, found 0.4% (2/455) risk for open abdomen treatment after EVAR and 0.9% (3/303) after open repair. This risk deference between the two groups may originate from a selection bias. Selection of more circulatory stable patients for EVAR results, however, in a lower incidence of IAH/ACS [103].

Whether to leave all patients with open abdomen as routine after open repair of a RAAA, or if it is better to close those patients who do not have a very tense abdomen, and follow them closely in the postoperative period, is still a controversial issue. The strategy of leaving many patients with open abdomen was first reported from the Mayo Clinic. They reported having left 19% open after AAA repair (43/223) [104]. A similar experience was reported from Zürich [96].

This controversy should be solved and addressed by randomized control trials. Based on the current knowledge, the updated consensus document favors primary closure and close IAP monitoring unless the abdomen is tense and difficult to close, which is the case in approximately 5–10% of patients.

5.5 Summary

Open abdomen and damage control philosophy are ill defined and studied in the acute non-trauma surgical setting. Although many surgeons practice this approach in diverse abdominal pathologies, no common denominator can be outlined in the reported cohort. Only well-conducted studies based on internationally agreed nomenclature will address the issue of who will be the patient that will benefit from the open abdomen and damage control strategy.

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The Open Abdomen in Trauma

6

Walter L. Biffl and Ernest E. Moore

In the 1980s, it became recognized that patient survival could be improved by limiting surgery to critical maneuvers including control of surgical hemorrhage and control of spillage from hollow visceral injuries [1–4]. This "damage control" approach was generally targeted at patients sustaining massive intra-abdominal trauma, with hypothermia, acidosis, and coagulopathy triggering a "bloody vicious cycle" that culminated in rapid demise [5]. The open abdomen (OA) in trauma typically is a component of damage control surgery (DCS). It is estimated that DCS techniques are currently employed in 10–15% of trauma laparotomies [6]. At the conclusion of damage control laparotomy, the abdomen is left open to prevent the abdominal compartment syndrome (ACS). While the OA is safe and effective at avoiding intra-abdominal hypertension, it is not a benign procedure. In this chapter, we will review the indications for OA in trauma. We will discuss the process of physiologic restoration and timing of return to the operating room, the management of patients with OA, and briefly review our preferred techniques for abdominal closure.

6.1 Indications for Open Abdomen in Trauma

The indications for the OA have been addressed in Chap. 5. Briefly, the primary indication for OA in trauma is DCS. The decision to abort laparotomy is usually related to recalcitrant coagulopathy and the bloody vicious cycle [5] but may be additionally influenced by other factors such as inadequate blood products, limited surgical expertise, or multiple casualties [7]. It is important to note that the decision

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to pursue DCS is often based on multisystem injuries and the most severe injuries may be extra-abdominal (e.g., extremity vascular injuries), of which immediate reconstruction may not be practical.

Other indications for OA in trauma include:

- Avoidance of intra-abdominal hypertension (IAH) and abdominal compartment syndrome (ACS), in the setting of massive hemorrhage and resuscitation with retroperitoneal edema and visceral swelling
- The need for second-look laparotomy to assess bowel viability following mesenteric vascular injury
- The need to remove visceral packing
- Management of gross peritoneal contamination not amenable to resolution at the first operation
- Treatment of secondary ACS after trauma
- Major abdominal wall tissue loss

6.2 Preparation for the Open Abdomen in Trauma

Once the decision is made to abort the operative procedure, preparations should be made for the OA. Surgical bleeding must be controlled. Bleeding solid organs and open retroperitoneal spaces should be packed and topical hemostatic agents used as necessary. Spillage from hollow viscera must be controlled. This may involve stapled or suture closure of bowel injuries and drainage of biliary or pancreatic lacerations. Intestinal injuries not amenable to rapid stapling or suturing (e.g., duodenal injuries) may be temporarily controlled by drains until definitive reconstruction is appropriate. Major vascular injuries may be temporarily shunted [8].

In order to more rapidly exit the OR, to preserve healthy fascia, and to avoid ACS, the fascia and skin should be left open after DCS [9]. There are multiple methods of temporary abdominal closure, as described in Chap. 10. Our preference is the "Vac-Pack" technique, performed similar to that described by Barker and colleagues (Fig. 6.1) [10]. A sterile polyethylene plastic sheet is fenestrated to allow egress of ascites fluid and then spread over the bowel and extending under the abdominal wall laterally to the paracolic gutters. A towel is placed over the top of the sheet, filling the wound and as an additional barrier to bowel protrusion. However, in the scenario of high risk for ongoing bleeding, the towel is omitted to facilitate direct observation of the peritoneal cavity. Two silastic drains are placed along the wound edges to evacuate the ascites fluid from the wound. These may be tunneled through the skin or laid on top of the skin and brought out at the cephalad aspect of the wound. Finally, the entire wound and a generous margin of skin are covered with an incise drape. We favor an iodophor-impregnated drape that is adhesive and contains some antibacterial properties. The silastic drains are connected to bulbs, and the bulbs are connected to continuous wall suction, as ascites is produced at a high level during resuscitation.

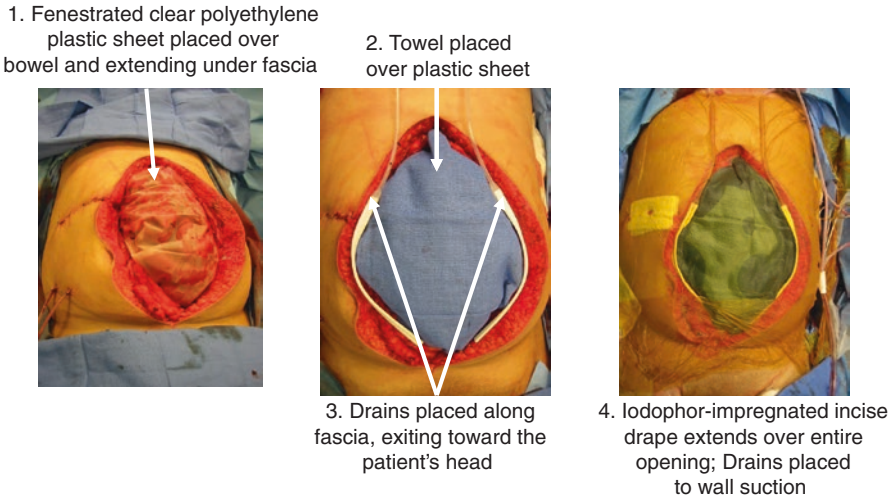
1. Fenestrated clear polyethylene plastic sheet placed over bowel and extending under fascia
2. Towel placed over plastic sheet
3. Drains placed along fascia, exiting toward the patient's head
4. Iodophor-impregnated incise drape extends over entire opening; Drains placed to wall suction

Fig. 6.1 "Vac-Pack" closure

6.3 Management of the Open Abdomen in Trauma

After initial DCS, the focus is on monitoring for complications. Ongoing bleeding can be occult, particularly if blood is contained deep within the abdomen and not reaching the drains. Frequent assessment of hemoglobin levels is important. Correction of coagulopathy is essential to limit hemorrhage and to restore physiologic normality prior to returning for definitive surgery.

In some cases, such as when a vessel is shunted, it is best to return the patient back to OR as soon as possible and within 6 h to avoid thrombotic complications [8]. In other cases, such as when a major liver injury is packed, it may be best to wait longer so that the packs may be removed with less chance of rebleeding. It is important to consider the indications.

Resuscitation should be targeted at correcting coagulopathy as well as reversing metabolic acidosis [7]. Continued bleeding or failure to correct acidosis may be indicators of uncontrolled surgical bleeding or ischemic viscera. These may be indications for returning to the OR sooner than was planned. Similarly, the development of IAH or ACS should prompt return to OR for exploration, hemorrhage control, and a temporary closure that accommodates additional bowel and retroperitoneal swelling.

The patient with prolonged OA is at risk of malnutrition, retraction of fascial edges, and risk for complications such as enterocutaneous fistula [11]. Nutritional support should be provided early, and the enteral route is preferred to enhance protein availability. Data from a multicenter prospective cohort study indicate that immediate enteral nutrition after damage control is safe, with no adverse effect on abdominal closure rate [12]. In addition, the investigators found a reduction in pneumonia associated with immediate enteral nutrition, consistent with previous work in injured patients.

6.4 Closure of the Open Abdomen in Trauma

In order to achieve fascial closure, the combination of negative pressure wound therapy with a dynamic fascial closure technique is optimal for achieving primary fascial closure [13]. Closure techniques are described in Chaps. 10, 17, and 18. In order to avoid later complications such as enteroatmospheric fistula, the bowel should be covered by 8 days. Our proposed technique has proven to have a very high fascial closure rate [14]. At the first reoperation, after definitive repairs are made, the fascia and skin are closed as long as there is no tension or significant increase in abdominal pressure. If complete closure cannot be attained, then negative pressure wound therapy is applied with white sponges on the bowel and fascial sutures providing traction (Fig. 6.2). A key element of the strategy is taking the patient to the OR every 48 h for further partial closure of the fascia as well as the skin (Fig. 6.3a, b).

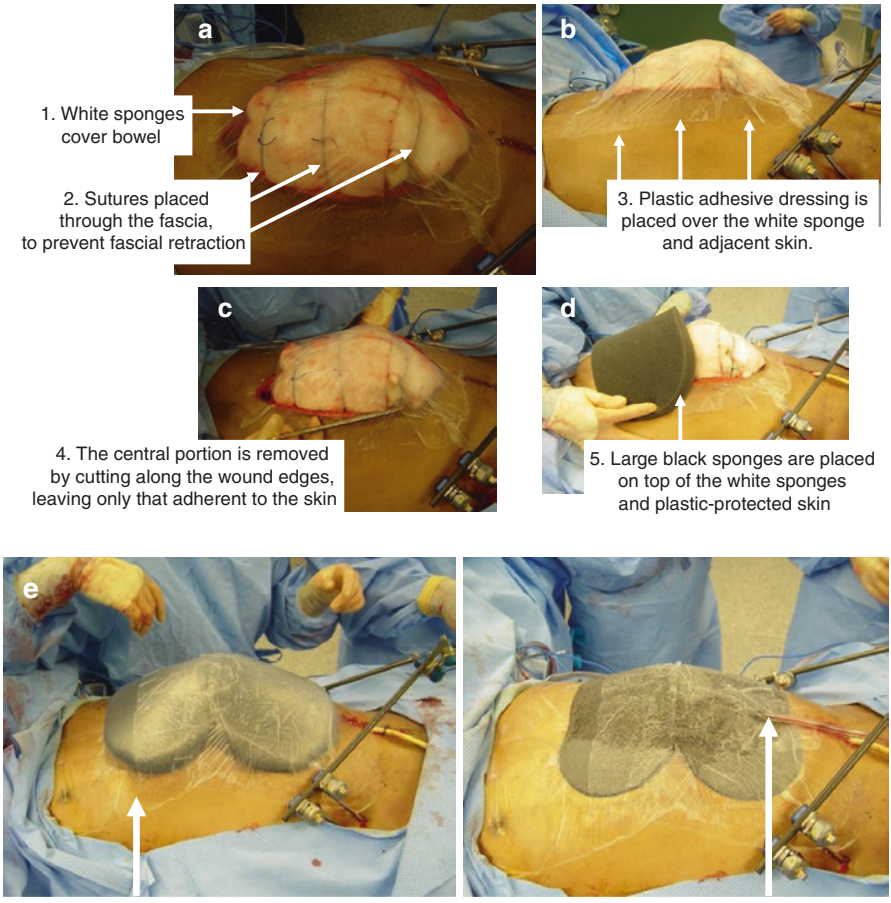
a
1. White sponges cover bowel
2. Sutures placed through the fascia, to prevent fascial retraction
b
3. Plastic adhesive dressing is placed over the white sponge and adjacent skin.
c
4. The central portion is removed by cutting along the wound edges, leaving only that adherent to the skin
d
5. Large black sponges are placed on top of the white sponges and plastic-protected skin
e
6. Black sponges are affixed with an occlusive dressing and standard suction tubing is placed.

Fig. 6.2 Negative pressure wound therapy with fascial suture traction

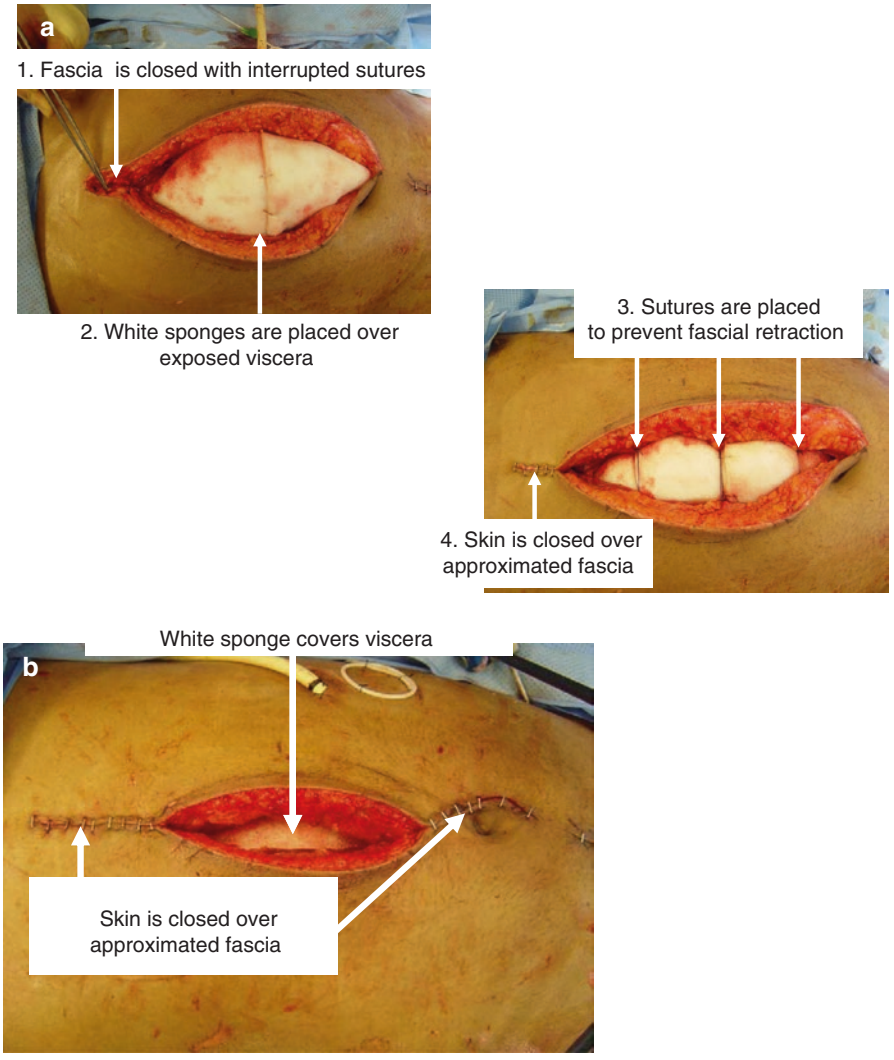
a
1. Fascia is closed with interrupted sutures
2. White sponges are placed over exposed viscera
3. Sutures are placed to prevent fascial retraction
4. Skin is closed over approximated fascia
White sponge covers viscera
b
Skin is closed over approximated fascia

Fig. 6.3 Sequential fascial closure

It is important to note that, while the OA approach as a component of DCS has saved many lives, it is not entirely benign. A prospective registry study of the American Association for the Surgery of Trauma found that large bowel resection, large-volume fluid resuscitation, and an increasing number of abdominal re-explorations were statistically significant predictors of enterocutaneous fistula, enteroatmospheric fistula, or intra-abdominal sepsis/abscess following DCS [15]. The group from Vanderbilt [16] found that 25% of patients had wound complications, and only 65% had primary fascial closure.

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Open Abdomen in Patients with Abdominal Sepsis

7

Massimo Sartelli, Federico Coccolini, Fausto Catena, and Luca Ansaloni

Key Points

- OA as part of a damage control strategy may be a lifesaving strategy in a well-selected group of surgical patients with severe abdominal sepsis.
- In critically ill patients with ongoing abdominal sepsis, the open abdomen may:
  - Allow early control of any persistent source of infection removing more effectively infected or cytokine-loaded peritoneal fluid.
  - Prevent abdominal compartment syndrome.
  - Defer definitive intervention and anastomosis until the patient is hemodynamically stable and thus better able to heal.
- Negative pressure therapy (NPT) should be used aiming to close the abdomen as soon as possible within 1 week.

7.1 Introduction

Abdominal sepsis is the host's systemic inflammatory response to bacterial or yeast peritonitis [2].

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Pathogenic microbial agents trigger cascades of events in sepsis by stimulating the host's immune system. Outer membrane component of gram-negative organisms such as lipopolysaccharide [LPS], lipid A, and endotoxin or gram-positive organisms such as lipoteichoic acid and peptidoglycan, as well as toxins from anaerobic bacteria or mannan from fungi, may stimulate macrophages and monocytes to produce tumor necrosis factor-α (TNF-α) and interleukin-1 and interleukin-6 (IL-1, IL-6), leading to the production of toxic mediators causing a complex, multifactorial syndrome that may evolve into conditions of varying severity and may lead to the functional impairment of one or more vital organs or systems.

Current treatment of severe sepsis involves prompt source control, early appropriate antimicrobial therapy, and adequate resuscitation.

Source control encompasses all measures undertaken to eliminate the source of infection, reduce the bacterial inoculum, and correct or control anatomic derangements to restore normal physiologic function. Its timing and adequacy are the most important issues in the management of intra-abdominal infections, because inadequate and late operation may have a negative effect on the outcome [3, 4].

In certain circumstances, infection not completely controlled may trigger an excessive immune response, and sepsis may progressively evolve [5].

Such patients may benefit from immediate and aggressive surgical treatment with subsequent relaparotomy strategies, to curb the spread of organ dysfunctions caused by ongoing sepsis.

In these patients, an early relaparotomy with surgical lavage of the peritoneal cavity and evacuation of toxic content and inflammatory cytokines may be crucial for stopping the septic cascade. This allows better control of the local inflammatory response and improved outcomes.

Three strategies in the management of these difficult patients have been reported [1]:

- Relaparotomy on demand (when required by the patient's clinical condition)
- Planned relaparotomy in the 36–48-h postoperative period (when relaparotomy is planned after the first operation)
- Open abdomen procedure

The OA concept is closely linked to damage control surgery and may be easily adapted to patients with ongoing sepsis.

The abbreviated laparotomy for trauma patients was defined as the initial control of surgical bleeding by simple operative lifesaving techniques. In the intensive care unit (ICU), subsequent resuscitation corrected hypothermia, acidosis, and coagulopathy. Once the patient had regained their physiologic reserve, definitive re-exploration and reconstructive surgery was performed with or without final abdominal closure [6]. This type of management may be applied for patients with severe abdominal sepsis.

Patients progressing from sepsis to septic shock can present with hypotension and myocardial depression associated with coagulopathy. These patients are

hemodynamically unstable and not optimal candidates for complex operative interventions.

After source control, abdominal closure may be temporary. In ICU patients, resuscitation is optimized and patients are stabilized. Over the following 24–48 h, when abnormal physiology is corrected, the patient can be safely taken back to the operating room for reoperation.

7.2 Relaparotomy

In critically ill patients with ongoing sepsis, OA approach may be required for different reasons including controlling any persistent source of infection, preventing abdominal compartment syndrome, and deferring definitive intervention and anastomosis.

Animal models have shown that peritonitis is associated with a significant and prolonged peritoneal inflammatory response [7]. The levels of selected peritoneal cytokines have been reported to be significantly different between animals that survived as compared to those who died following a septic challenge [8].

The reduction of the local inflammatory response can be best achieved with mechanical surgical control by reducing the load of cytokines and other inflammatory substances [9] and by preventing their production, thus removing the source itself. In some patients, more laparotomies are required to complete source control, and OA allows the surgeon to perform subsequent planned laparotomies more efficiently.

The final decision to perform a reoperation on a patient is based on the patient generalized septic response and on the lack of clinical improvement during early postoperative period [10, 11].

However, these conditions are not well defined [12] and often relaparotomy may be performed too late. In patients with ongoing sepsis, OA allows easy second look to control the source of infection and evacuate inflamed and toxic content.

7.3 Prevention of Abdominal Compartment Syndrome

The systemic inflammatory response syndrome, increased vascular permeability, and aggressive crystalloid resuscitation predispose to fluid sequestration with the formation of peritoneal fluid. These changes associated with forced closure of the abdominal wall may result in increased intra-abdominal pressure (IAP) leading to intra-abdominal hypertension (IAH) and abdominal compartment syndrome (ACS).

ACS is a potentially lethal complication on splanchnic, cardiovascular, pulmonary, renal, and central nervous systems.

Especially in patients with severe peritonitis, the physiological effect of ACS to gastrointestinal tract may aggravate the abdominal sepsis.

The earliest and most effective means of treating ACS in high-risk patient may be surgical decompression. Presumptive decompression should be considered at the

time of laparotomy in patients who demonstrate risk factors for ACS [1]. The decision to perform a laparostomy in patients with abdominal sepsis should be always based on the intraoperative judgment of the surgeon.

7.4 Delayed Intestinal Anastomosis and Definitive Intervention

An additional advantage of the OA strategy in abdominal sepsis is to delay the bowel anastomosis [12] in patients hemodynamically stable and thus unable to heal.

In patients with severe secondary peritonitis and significant hemodynamic instability and compromised tissue perfusion, primary anastomosis is at high risk for anastomotic leakage resulting in increased mortality.

In these patients, consideration should be given to initially control the source of peritoneal contamination and delay the bowel anastomosis [13].

7.5 Fascial Closure

Following re-exploration, the goal is early and definitive closure of the abdomen, in order to reduce the complications associated with an open abdomen.

Early definitive closure is defined as fascial abdominal closure achieved less than 7 days after the initial OA procedure. It allows to prevent or reduce the risk of these complications [14, 15].

Delayed fascial closure is defined as fascial abdominal closure achieved 7 or more days [16, 17].

In order to evaluate whether early fascial abdominal closure had advantages over delayed approach, a systematic review and meta-analysis was published in 2014 [18].

The study confirmed the clinical advantages of early fascial closure compared with delayed closure in the treatment of patients with open abdomen.

In patients with delayed abdominal wall closure, fascial edges lateralization may lead to unfavorably high tensile midline forces, and fascial closure may be more problematic. In these patients, abdominal wall closure should approximate the fascial edges progressively and incrementally, each time the patient should undergo surgery until it is completely closed.

The ideal temporary abdominal closure (TAC) method should protect the abdominal contents, prevent evisceration, allow removal of infected or toxic fluid from the peritoneal cavity, prevent the formation of fistulae, avoid damage to the fascia, preserve the abdominal wall domain, make reoperation easy and safe, and facilitate definitive closure [1].

Several materials can be used to achieve temporary closure of the abdomen: gauze, mesh, impermeable self-adhesive membrane dressings, zippers, and negative pressure therapy (NPT) techniques.

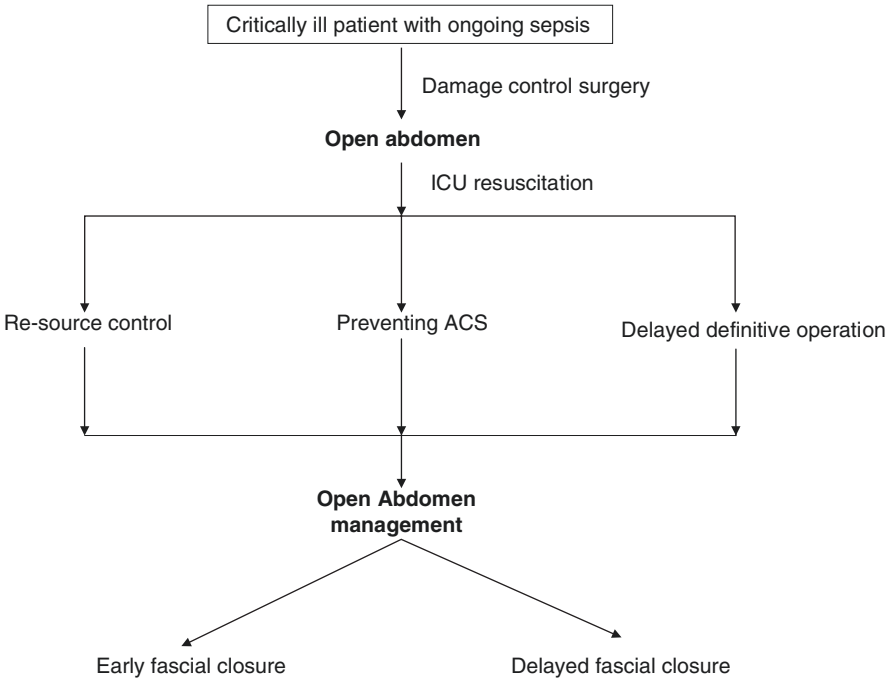

Fig. 7.1 Flowchart for the open abdomen procedure in patients with ongoing abdominal sepsis

A systematic review and meta-analysis of the open abdomen and temporary abdominal closure techniques in non-trauma patients was published in 2015 [19]. The overall quality of the included studies was low, and the indications for open abdominal management differed considerably. Negative pressure therapy (NPT) was the most frequently described TAC technique (38 of 78 series).

The highest weighted fascial closure rate was found in series describing NPT with continuous mesh or suture mediated fascial traction and dynamic retention sutures. Weighted rates of fistula varied from 5.7% after NPT with fascial traction, 14.6% for NPT only, and 17.2% after mesh inlay.

In Fig. 7.1, the flowchart for the open abdomen procedure in patients with ongoing abdominal sepsis is illustrated.

Conclusions

Outcomes of complicated intra-abdominal infections accompanied by severe sepsis are related to early diagnosis, early surgical management with source control, aggressive critical care management, and aggressive and early optimization of physiology.

OA as part of a damage control strategy may be a lifesaving strategy in selective patients with severe abdominal sepsis. Once severe sepsis has been controlled, definitive surgical reconstruction should be performed as soon as possible.

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Open Abdomen in Acute Pancreatitis

8

Ari Leppäniemi

Key Points

- Monitor and treat increased intra-abdominal pressure in severe acute pancreatitis.
- Early midline laparostomy is required in abdominal compartment syndrome resistant to nonoperative management including percutaneous drainage of pancreatic ascites.
- Mesh-mediated vacuum-assisted closure is the safest and most effective temporary abdominal closure technique in open abdomen.
- Leaving the abdomen open as part of surgical necrosectomy is not warranted.

8.1 Introduction

While in the majority of patients acute pancreatitis is a mild, self-limiting disease, about 15% of the patients develop severe disease defined by the development of persistent organ failure [1]. The mortality in acute pancreatitis is mainly associated with multiple organ failure (MOF), whereas the risk of dying is minimal in patients with no or transient organ dysfunction [2–4]. MOF develops early during the course of acute pancreatitis, over half of the patients with severe acute pancreatitis (SAP) have signs of organ dysfunction on hospital admission, and most of the organ dysfunctions develop within the first 4 days after admission [3, 5]. Over half of the deaths occur within the first week from onset of the disease, and deaths usually occurred within a week after manifestation of MOF [6]. Treatment modalities of

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MOF are supportive including fluid replacement therapy, vasopressors, mechanical ventilation, and renal replacement therapy when necessary.

8.2 Abdominal Compartment Syndrome

Abdominal compartment syndrome (ACS) may aggravate MOF, and therefore, monitoring of intra-abdominal pressure (IAP) is crucial for identification of patients at risk of ACS [7]. Development of ACS should be prevented and if possible treated by conservative methods including percutaneous drainage of pancreatic ascites, if present in large amounts confirmed by ultrasound examination. Surgical decompression is the last but the most effective way to decrease IAP and should not be postponed too late if patient has developed ACS [8, 9].

8.3 Infected Pancreatic Necrosis

Patients with acute pancreatitis have a considerable risk for developing secondary infections including bacteremia, pneumonia and infection of pancreatic or peripancreatic necrosis. Extrapancreatic infections occur predominantly during the first week of illness, whereas pancreatic necrosis becomes infected later [10]. Organ failure, early bacteremia, and the extent of pancreatic necrosis are associated with increased risk of infected necrosis. Surgical necrosectomy is the last resort if more conservative management including percutaneous drainage fails [11]. The mortality is very high in patients with persistent organ failure complicated with infected pancreatic necrosis [12].

8.4 Open Abdomen

While open abdomen by itself is a morbid procedure and should not be used lightly, there are two clinical conditions where open abdomen can be used as a treatment strategy in patients with SAP, either to treat intra-abdominal hypertension (IAH) and ACS or as a method to treat infected pancreatic necrosis.

While IAH is present in the majority of patients with SAP treated in an intensive care unit (ICU), ACS develops in a fraction of them, usually within the first few days and associated with excessive fluid resuscitation [7, 13]. Recent studies show also that intra-abdominal ischemia is common in SAP patients with ACS [14].

8.5 Surgical Decompression

Surgical decompression has a marked effect on organ function, especially among subsequent survivors [9, 15]. The most common technique for surgical decompression is the vertical full-thickness midline laparostomy, but other alternatives such as transverse laparostomy or subcutaneous linea alba fasciotomy have been described. Currently the most widely used is the midline laparostomy that has been showed to decrease IAP

effectively [16]. It is rapid and easy to perform, but it is associated with a high risk of intestinal fistulae and in many cases failure to close the fascia requiring complex reconstructive surgery at a later stage. Transverse laparostomy is a promising alternative, and isolated reports have shown its effectiveness in reducing IAP [17]. Although it takes slightly longer to perform than midline laparostomy, same principles of managing the open abdomen can be applied without additional equipment. The major disadvantage could be the loss of abdominal and back extensor muscle functions, if fascial closure could not be achieved. A third alternative used in SAP is the subcutaneous linea alba fasciotomy, where the fascia is incised through three small skin incisions leaving the rest of the skin and the peritoneum intact [18]. Although it eliminates the open abdomen, it might not be always effective enough [19]. In addition, the subcutaneous fasciotomy always results in a ventral hernia requiring repair later on.

Although many temporary abdominal closure techniques (TAC) are in use, the mesh-mediated vacuum-assisted closure seems to have the highest fascial closure and lowest enteric fistula rates [20–23] (Figs. 8.1, 8.2, 8.3, 8.4, 8.5).

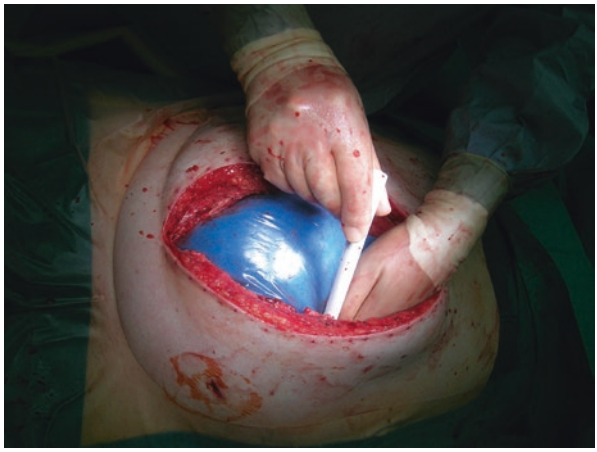

Fig. 8.1 Applying the inner plastic layer

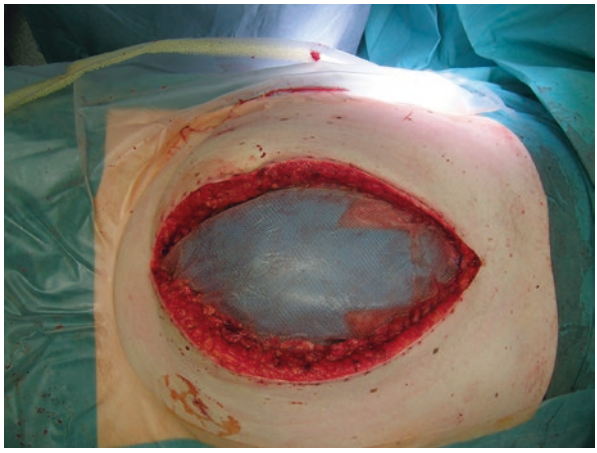

Fig. 8.2 Mesh sutured to the fascia over the underlying plastic sheath

Fig. 8.3 Sponge and covering air-tight plastic applied to create negative pressure

Fig. 8.4 After several rounds of tightening the mesh at dressing changes, the gap is sufficiently narrow to allow mesh removal and fascial closure

Fig. 8.5 Delayed fascial closure

8.6 Necrosectomy

With the new definitions of pancreatic necrosis, acute necrotic collection (ANC) (Fig. 8.6), and walled-off necrosis (WON) (Fig. 8.7) and better understanding of the natural progress of the disease process, the management of infected pancreatic necrosis has undergone a shift toward a more conservative approach as well as postponing surgical intervention well after 4 weeks of the onset of the symptoms [1, 9, 10, 24]. Although in the past, the open abdomen strategy was offered as an

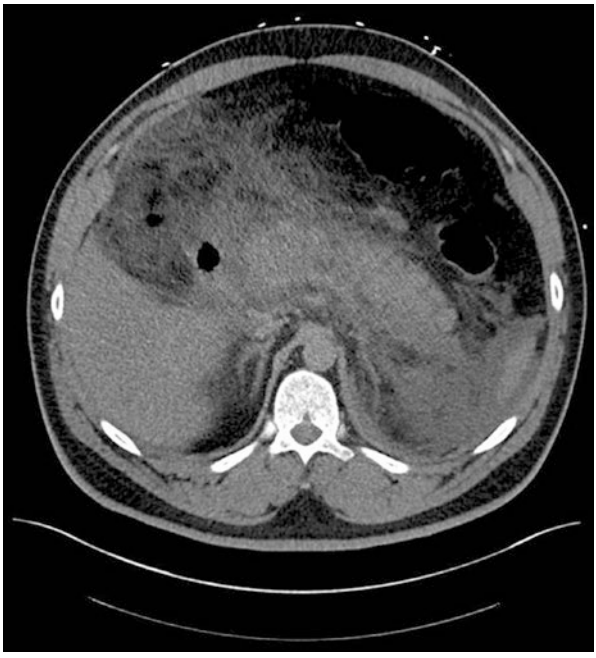

Fig. 8.6 CT image of acute necrotic collection (ANC)

Fig. 8.7 CT image of walled-off necrosis (WON)

alternative to closed drainage after open necrosectomy, retrospective studies have shown that the results of the open abdomen strategy are poor compared with closed and minimally invasive procedures [25].

Take-Home Messages

The use open abdomen in patients with severe pancreatitis should be limited to the management of ACS. Surgical decompression should be used only if nonoperative management fails, but when needed, it should be performed promptly. Using an adequate temporary abdominal closure technique, such as the mesh-mediated vacuum-assisted closure, high fascial closure and low enteric fistula rates can be achieved.

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The Open Abdomen in Non-traumatic Vascular Emergencies

9

S. Acosta, A. Wanhainen, and M. Björck

9.1 Introduction

Open abdomen treatment is sometimes necessary to save lives in trauma and non-trauma surgical conditions [1]. The vascular surgeon needs to be aware of the indications for open abdomen therapy (see Chap. 5), zones of injury and exposure of major abdominal vessels (see Chap. 6), and preferable temporary abdominal closure methods in vascular surgery. Besides damage control laparotomy [2] and short-term open abdomen therapy after major vessel trauma with severe bleeding and associated intra-abdominal injuries in the often younger patients, long-term open abdomen therapy is also necessary in the prevention and treatment of abdominal compartment syndrome (ACS) in elderly following repair of ruptured abdominal aortic aneurysms (AAA). Damage control laparotomy or abbreviated laparotomy with second-look laparotomy may also be an advantageous strategy in the management of patients with abdominal catastrophes such as advanced stages of acute mesenteric ischemia (AMI). This chapter deals only with open abdomen in non-traumatic vascular emergencies.

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9.2 Open Abdomen Following Repair of Ruptured AAA

9.2.1 Abdominal Compartment Syndrome Following Ruptured AAA

Decompressive laparotomy due to abdominal compartment syndrome (ACS) following open or endovascular repair of ruptured AAA (RAAA) is performed increasingly often. The proportion of patients who develop ACS after open and endovascular aneurysm repair (EVAR) of ruptured abdominal aortic aneurysm (AAA) has been reported to be around 30% [3] and 20% [3, 4], respectively. Increased blood loss, hypotension, massive fluid resuscitation, and prolonged occlusion of the aorta with subsequent reperfusion injury with viscera edema lead to increased risk of intra-abdominal hypertension or ACS. Perioperative adherence to massive transfusion protocols with 1:1:1 ratio of units of packed red blood cells and plasma to platelets may decrease the risk of ACS in massive uncontrolled hemorrhage [5]. If identified early postoperative, intra-abdominal hypertension can be treated with epidural analgesia, neuromuscular blockade, and diuretics. Such a proactive approach can prevent the development of ACS. When ACS is identified late or when the intra-abdominal pressure reaches 30 mmHg, emergent decompression laparotomy is necessary to save life [6]. Recognition and timely management of ACS after AAA repair is crucial for improvement of survival and development of intestinal ischemia during ACS is associated with increased mortality [7].

9.2.2 When Deciding to Leave the Abdomen Open

The frequency of patients with RAAA in series reporting damage control laparotomy in non-trauma patients varies between 5 and 25% [8–11] (Table 9.1). In one report of 13 patients, the abdomen was temporarily closed by vacuum pack technique, with or without retroperitoneal packing after repair of RAAA, in order to prevent ACS. This damage control management with early decision in leaving

Table 9.1 Frequency of vascular diseases in series of damage control laparotomy for non-trauma patients

First author (year)	Country	Vascular disease (%)	Ruptured AAA (%)	Acute mesenteric ischemia (%)
Finlay (2004) [8]	United Kingdom	4/12 (33)	3/12 (25)	–
Freeman (2005) [29]	Australia	20/20 (100)	–	20/20 (100)
Person (2009) [30]	Israel	10/31 (31)	–	10/31 (31)
Subramanian (2010) [31]	USA	13/88 (13)	–	13/88 (13)
Tadlock (2010) [9]	USA	13/13 (100)	13/13 (100)	–
Goussous (2013) [10]	USA	30/111 (27)	5/111 (5)	25/111 (23)
Khan (2013) [11]	New Zealand	15/42 (36)	2/42 (5)	13/42 (31)

the abdomen open resulted in earlier ICU arrival for intense resuscitation of profound shock, persistent coagulopathy, acidosis, and hypothermia and a mortality of 54% [9].

The decision to leave the abdomen open may also be taken at the end of the operation: The abdomen may simply be left open since the abdomen may be impossible to close due to, for instance, excess of fluid infusion perioperatively and swelling of the abdominal compartment. Sometimes, the surgeon starts to suture the fascial edges together but removes the suture, since closure would have resulted in a too tense abdomen and the risk of developing ACS. Fascial closure followed by on-table intra-abdominal pressure measurement may also result in removal of the fascial suture to allow full abdominal decompression. The abdomen is more often reopened after open repair at a second operation due to development of secondary ACS. Decompressive laparotomy may be performed directly or as a second operation after EVAR. Relaparotomy after open repair and laparotomy after EVAR may also be performed if intestinal, usually colonic, ischemia develops, a condition that coexists with ACS [12].

9.2.3 Prolonged Open Abdomen Therapy May Be Necessary

The elderly patients with AAA often have, in contrast to younger trauma patients, coexistent comorbidities and compromised physiological functions such as cardiac, pulmonary, and renal insufficiency [13], leading to a relative therapeutic resistance to reduce the fluid overload that usually arises after an emergency repair of RAAA. Thus, the duration of open abdomen therapy is often prolonged, before abdominal closure is possible. It is therefore very important to have a durable dressing system that minimizes the risk of further complications and facilitates complete fascial closure.

9.2.4 Temporary Abdominal Closure in AAA Repair

In a recent systematic review, the most effective TAC was the vacuum-assisted wound closure and mesh-mediated fascial traction (VACM; Fig. 9.1) after AAA repair [14]: The primary fascial closure rate varied from 96 to 100% in three studies [15–17]. The median time to closure of the open abdomen in these three studies ranged from 10.5 [16], 17 [15] to 36 [17] days. No patient with long-term open abdomen therapy with the technique was left with a planned ventral hernia. The graft infection rate was 0% [16, 17] to 6.7% [15]. The reported graft infection rate after vacuum pack treatment has also been reported to be very low, 0% [9, 18] to 7.1% [19]. In the prospective study [15] referred to above, 2 out of 30 patients developed enteroatmospheric fistulae after open abdomen therapy with the VACM method. Both had an underlying intestinal ischemia, underwent bowel resections, and died. Incisional midline hernia occurred in 9 of 15 patients at 1-year follow-up with computed tomography. Three (20%) incisional hernias were symptomatic, of which two were repaired.

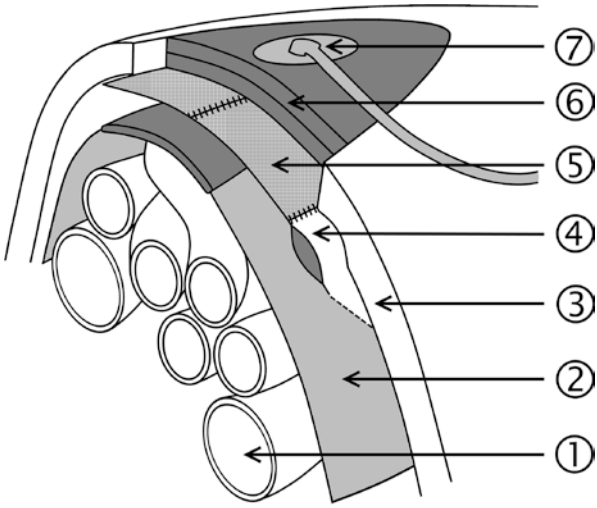

Fig. 9.1 Vacuum-assisted wound closure and mesh-mediated fascial traction technique: 1, bowel; 2, visceral protective layer; 3, abdominal wall; 4, abdominal wall fascia; 5, polypropylene mesh; 6, two pieces of polyurethane foam placed on top of the mesh and subcutaneously between the wound edges; 7, tubing set with an interface pad attached to an opening in the self-adhesive drapes and connected to the vacuum source

The VACM is as a good alternative for the treatment of a clean, Grade IA, open abdomen after AAA repair [20]. The method facilitates high primary fascial closure rates even after several weeks of open abdomen therapy since this combined technique with NPWT and mesh-mediated fascial traction counteracts lateral retraction of the fascia. Achievement of high fascial closure rates with this technique have been reproduced in general surgery patients [21–25]. As soon as there are intestinal and/or infectious complications, affecting the grading of the open abdomen, the primary delayed fascial closure rate decreases, and the risks of intestinal fistulae and graft infections increase [26]. The duration of open abdomen treatment seems to be a crucial factor, since most severe complications usually develop after 2 weeks [27].

9.3 Open Abdomen Following Management of Acute Mesenteric Ischemia

The increasing use of revascularization of the mesenteric arterial circulation in acute mesenteric ischemia has created an increased demand of second- or third-look laparotomies for assessing bowel viability and definitive reconstructions of the bowel [28]. In patients that develop peritonitis, a damage control strategy is sensible [29]. The frequency of patients with acute mesenteric ischemia in series reporting damage control laparotomy in non-trauma patients varies between 13 and 31% [10, 11, 30, 31] (Table 9.1), which means that acute mesenteric ischemia is a rather common cause when performing damage control laparotomies. Survival rate was reported to be 62% in 16 actively treated patients in one series [29]. Collaboration between vascular and general surgeons may improve outcome. In

the recently developed *European Society of Vascular Surgery Guidelines on the management of diseases of the mesenteric arteries and veins*, damage control surgery and second-look laparotomy are recommended when bowel infarction has developed in patients undergoing intestinal revascularization [32]. This strategy is less well studied in non-occlusive mesenteric ischemia and mesenteric venous gangrene [33], but liberal use of second-look laparotomy seems to be a rational approach in these entities of acute mesenteric ischemia as well.

9.3.1 Treatment of Acute Superior Mesenteric Artery Occlusion

Optimal treatment may include both open and endovascular surgery, and patients are best treated in a vascular center with a hybrid operating room, if available within a reasonable distance. From preoperative clinical and CT angiography evaluation, it can be determined whether the patient has peritonitis or not and whether the occlusion is embolic or thrombotic. Laparotomy is indicated if there is peritonitis to evaluate the extent and severity of intestinal ischemia by inspection of color of the intestines, dilatation and peristaltic motion of the bowel, visible pulsations in the mesenteric arcade arteries, and bleeding from cut surfaces. Acute mesenteric arterial revascularization, open, hybrid, or endovascular, is done preferably before any bowel surgery (Fig. 9.2). There is rarely any indication for revascularization of both the SMA and the coeliac trunk, of which SMA revascularization seems to be more important and durable. In an acute thrombotic occlusion, retrograde open mesenteric stenting [34] may be a very useful hybrid approach where SMA is exposed at laparotomy, followed by retrograde recanalization of the thrombotic and atherosclerotic occluded SMA, and stented in retrograde or antegrade manner (Fig. 9.3). All initial emergency vascular and necessary visceral surgery should be performed during the same operation.

9.3.2 Temporary Abdominal Closure in Acute Mesenteric Ischemia

Even a low grade of intra-abdominal hypertension may become fatal when a large portion of the intestinal tract is gangrenous [27]. In this situation it is important to optimize the perfusion of the remaining bowel, which often requires open abdomen treatment [35]. In the event of bowel perforation, the affected intestinal segment is rapidly resected, followed by mesenteric revascularization, leaving the reconstruction of the intestinal tract until second-look laparotomy after 18–36 h. The optimal TAC method in patients with acute mesenteric ischemia is not clear. While temporary skin-only closure may be a good option in some patients, full open abdomen to optimize visceral circulation may be necessary in others. In some reports, long-term NPWT in open abdomen due to secondary peritonitis [36] or intestinal ischemia requiring bowel resection and anastomosis [25] were factors

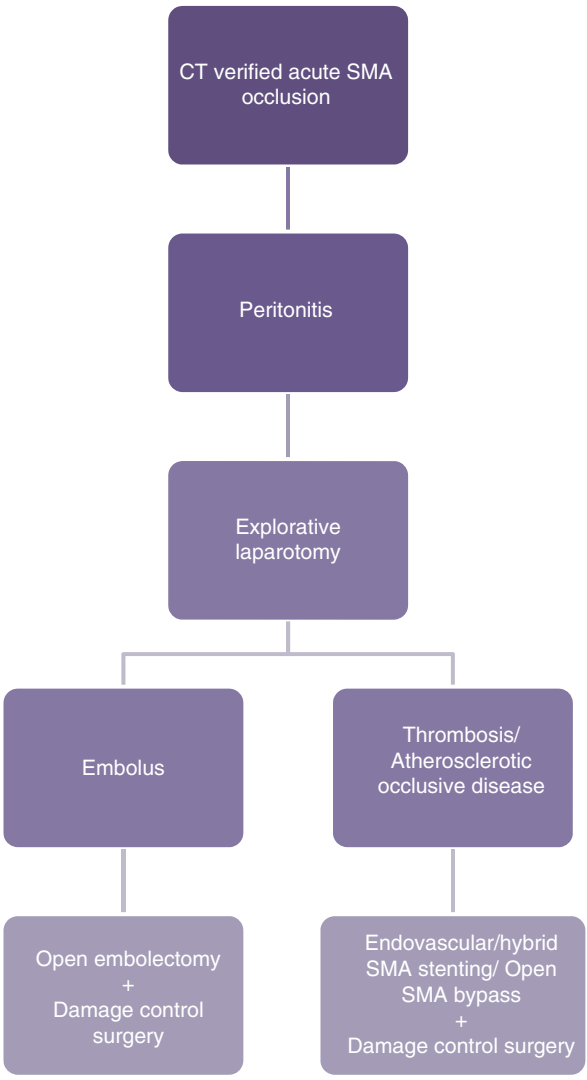
CT verified acute SMA occlusion
Peritonitis
Explorative laparotomy
Embolus
Thrombosis/ Atherosclerotic occlusive disease
Open embolectomy + Damage control surgery
Endovascular/hybrid SMA stenting/ Open SMA bypass + Damage control surgery

Fig. 9.2 Flowchart in patients with acute SMA occlusion and peritonitis

associated with the development of enteroatmospheric fistulae and high mortality. Of note, these studies did not have a control group. In a recent systematic review and meta-analysis [37] of the open abdomen and TAC techniques in non-trauma patients, NPWT with continuous fascial traction had higher delayed fascial closure rate and lower enteroatmospheric fistula formation rate compared to NPWT alone, emphasizing the importance of having an effective method to close the abdomen as quickly as possible, to reduce the risks of complications associated with an open abdomen.

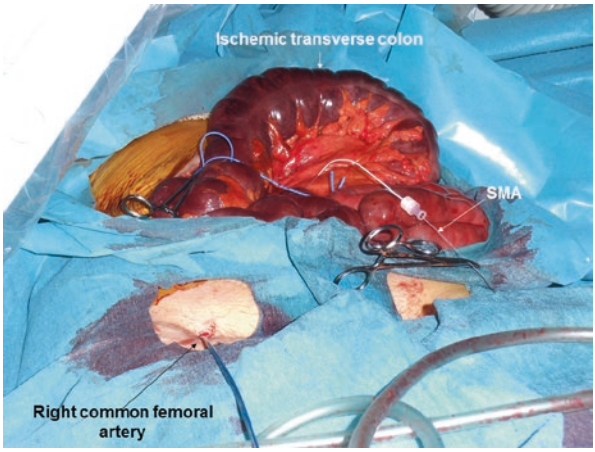

Fig. 9.3 Retrograde open mesenteric stenting: A puncture is made in the main trunk of the SMA with a micropuncture needle, and the occlusion is often recanalized easily with a guidewire into the aorta. The proximal SMA atherosclerotic occlusion is then crossed with a stiff catheter, exchanging for a hydrophilic guidewire. This wire is snared in the aorta using a snare passed through a femoral access and then brought out, creating through-and-through access. An introducer is placed antegrade in the SMA over the through-and-through wire from the femoral artery, followed by stenting

9.3.3 Delayed Bowel Anastomosis or Stoma

At first or second look, bowel resection is performed without reconstruction. At the following laparotomy, bowel anastomosis or stoma is created. In one center, a temporary stoma is created, followed by administration of parenteral nutrition until the surgical recovery phase is over [38]. In patients undergoing extensive bowel resections, proximal resection of the jejunum, or multiple resections, bowel anastomosis after effective intestinal revascularization may be beneficial in avoiding high output stoma, short bowel syndrome, and the increased mortality associated with intestinal failure [39].

Take-Home Messages

- Recognition and timely management of abdominal compartment syndrome after repair of ruptured abdominal aortic aneurysm is crucial for improvement of survival.
- Vacuum-assisted wound closure and mesh-mediated fascial traction is a good temporary abdominal closure method in patients requiring prolonged open abdomen therapy.
- Patients with acute superior mesenteric artery occlusion and peritonitis should undergo damage control laparotomy and, preferably, intestinal revascularization first.

Conflict of Interest Statement The authors declare no conflict of interest.

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The Management of the Open Abdomen: The Temporary Closure Systems

10

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Key Points

- NPWT with continuous fascial traction is suggested as the preferred technique for TAC.
- TAC without NPWT (e.g., mesh alone, Bogota bag) whenever possible should not be applied for the purpose of TAC, because of low delayed fascial closure rate and being accompanied by a significant intestinal fistula rate.
- Wittman patch could be safety and less expensive alternative.

10.1 Background

The surgeon can use different techniques to manage an open abdomen (OA). The techniques reported in the literature have the advantage of being diverse and applicable in all the countries. Some techniques are easy to apply and cheaper and could be used also in countries with a lower economic status. Others techniques are more expensive and are developed in rich countries. However in our era, the attention to a spending review meant that even these countries researched cheaper but equally effective devices [1, 2].

The most important difference between devices and techniques is to apply or not a negative pressure therapy. The first techniques as towel clip closure, skin running

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suture, Bogota bag, zipper, and sheet temporary closure leave the abdomen open by protecting the bowel or close temporarily the abdomen but do not apply any negative pressure and do not remove any fluid on the abdomen cavity. The recent techniques develop a system with a negative pressure to reduce fluids in the abdomen through aspiration drainages or aspiration continuous or intermittent pump.

The other important point to take into consideration is the pathology underlying the choice of the open abdomen management [3]. A different technique could be used in young trauma patients compared to septic elderly patients or to severe acute pancreatitis patients [3]. In fact different pathophysiological mechanisms underlie these clinical conditions, and a different approach can be used.

However a best device that can achieve a good fascial closure preserving the abdominal wall domain of the intra-abdominal organs is not found yet [1].

10.2 Not Vacuum-Assisted Techniques

10.2.1 Skin-Only Closure Techniques (Towel Clips, Skin Running Suture)

These two techniques which are based on approaching skin with towel clips or running sutures are almost abandoned. They consist in closing the skin only by making the edges closer using towel clips (Fig. 10.1) or rapid monofilament running suture [2, 4, 5]. They are used to perform temporary abdominal closure (TAC) or open abdomen in quickly procedures as in damage control surgery (DCS) especially in trauma [1, 2, 4, 6]. These techniques are cheap, immediately available, and easy to apply also for non-expert surgeons [7]. However, the main disadvantage is that closing the abdomen, also if temporary, could increase the intra-abdominal pressure (IAP), especially in the case of the running suture where ACS could occur in 13–36% [1, 7]. Other problems are the impossibility to assess the intra-abdominal

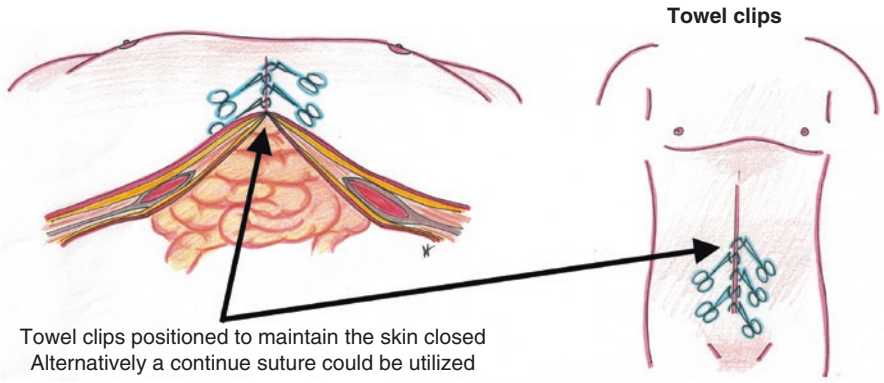


Fig. 10.1 Towel clips (authorized reproduction from www.clinicalregisters.org)

content without reopening the abdomen, the impossibility to remove the infected fluids or toxin- and cytokine-rich intraperitoneal fluids, and the impossibility to prevent edge retraction [4].

10.2.2 Bogota Bag

The "Bogota bag," so named by Mattox after observing the usage of intravenous bag in Bogota, Colombia, or silo is a sterile large intravenous fluid bag (3 l irrigation bag) that is sutured to the skin or to the fascia (Fig. 10.2) [1, 2, 4]. Other variants could be bowel bag, Steri-Drape, and Silastic cloth [5]. This simple and cheap technique, immediately available, could be lifesaving. Moreover, the transparency of the bag allows seeing inside the viscera. The other advantage is that by leaving the skin and the fascia open, it is possible to prevent or treat the intra-abdominal hypertension (IAH) and the abdominal compartment syndrome (ACS) [2]. As the abovementioned techniques, the "Bogota bag" does not allow to remove intra-abdominal fluids and toxins and does not allow to reduce visceral edema [1, 4]. The non-application of a negative pressure could explain the low rate of enterocutaneous fistula reports (0–14.4%). On the other hand, no retraction of the fascia is performed (definitive fascial closure rates lower than 28%) [7].

Some centers, as the Puerto Rico Trauma Center (PRTC) [8], use a modified Bogota bag approach by using dynamic-like retention sutures. They performed a small incision 1 cm away from the surgical incision margins, intravenous tubes were inserted through the incisions, and suction drains were inserted bilaterally near the skin margins above the internal sterile bag to remove fluids. An external smaller sterile bag was sutured to the skin. The intravenous tubes are stretched every 24–36 h to re-approximated the abdominal wall. That approach reaches an abdominal closure near to 91%. The in-hospital mortality report in the literature is 12% [5].

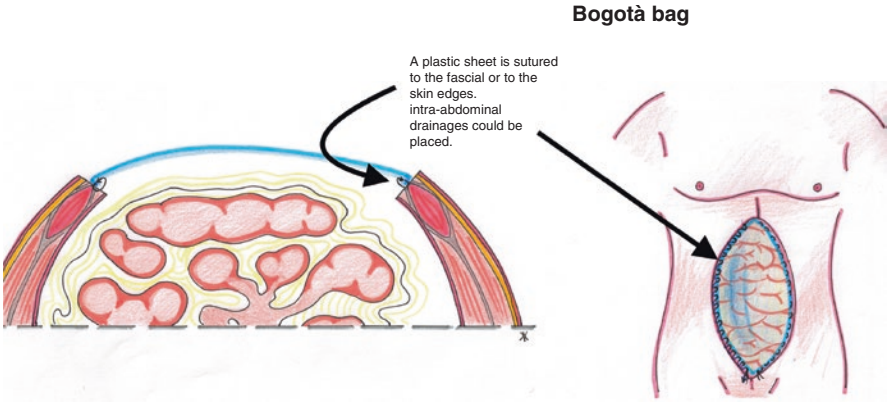


Fig. 10.2 Bogota bag (authorized reproduction from www.clinicalregisters.org)

10.2.3 Frequent Assessment of Abdominal Content (Fastac), Absorbable or Nonabsorbable Mesh, and Sheet Temporary Closure

In the mid-1980s–2000s, absorbable or nonabsorbable meshes or sheets were used to make the re-exploration of the abdominal cavity easier [4, 8]. The nonabsorbable meshes can be sutured at the fascia creating a tension-free closure and allowing a gradual re-approximation of the fascia when the mesh/sheet is plicated reducing the abdominal defect (Fig. 10.3) [2]. At the re-exploration, the mesh/sheet can be cut in the middle and after re-sutured approaching the two edges (also associated with a negative pressure therapy to increase the primary fascial closure rates) [2, 4]. The rates of primary closure ranged from 33 to 89% in case of use of nonabsorbable meshes [5]. However, due to the high risk of development of enterocutaneous fistula (6.6–14.7% to 75% in other series), the nonabsorbable meshes/sheets need to be removed and eventually substituted with biologic meshes [4]. Nevertheless, Scholtes et al. [9] in a recent article report a reduced rate of incisional hernia also in patients with contaminated or dirty abdomen with an overall mortality rate of 8% and a enterocutaneous fistula rate of 22% not influenced by the use of mesh. However, the authors report some bias due to the retrospective analysis and the indications for mesh implantation. The presence of porous in these meshes could be an advantage to facilitate the drain of intra-abdominal fluids. However, the use of PTFE microporous mesh seems to increase the risk of intra-abdominal infections [5].

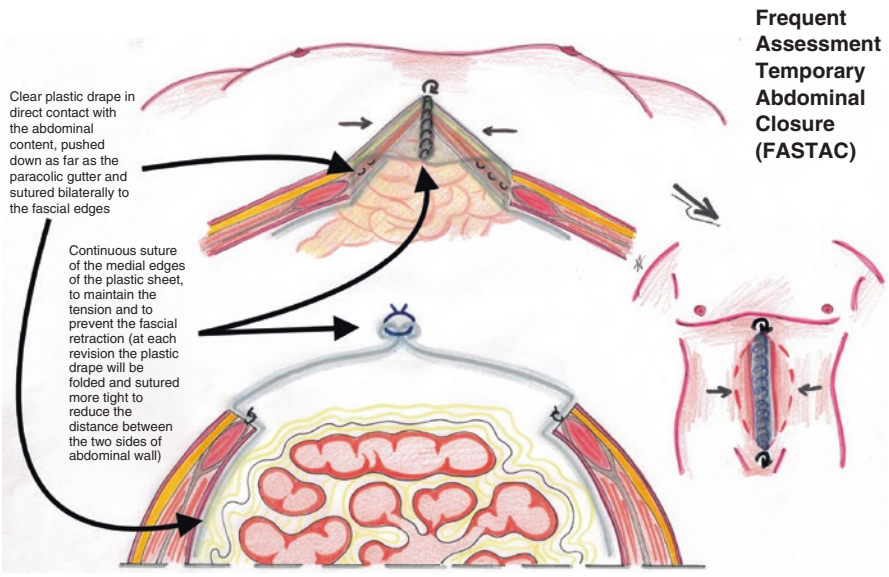

Frequent Assessment Temporary Abdominal Closure (FASTAC)
Clear plastic drape in direct contact with the abdominal content, pushed down as far as the paracolic gutter and sutured bilaterally to the fascial edges
Continuous suture of the medial edges of the plastic sheet, to maintain the tension and to prevent the fascial retraction (at each revision the plastic drape will be folded and sutured more tight to reduce the distance between the two sides of abdominal wall)

Fig. 10.3 FASTAC (authorized reproduction from www.clinicalregisters.org)

The absorbable meshes (as Vicryl mesh or Dexon mesh) can be left in place; however, they showed less tension above the fascial edges increasing the risk of incisional hernia [1, 4, 5, 7]. The risk of enteroatmospheric fistula, when the mesh is placed in contact with the bowel, is 5–10% for absorbable meshes [5]. In 2013 Sutton et al. [10] report a case of the use of Gore Bio-A mesh, a biocompatible synthetic polymer that is absorbed in 6 months. This mesh is cost-effective, feasible, and safe also in contaminated field and helps the growth of granulation tissue [10]. Meshes can be also used to increase granulation tissue formation in patients in which skin closure is not possible, to allow the substrate for a skin graft later [1, 4, 5].

10.2.4 Zipper

The zipper consists in closing the skin or the fascial layer only by moving the edges close by positioning a common zipper sutured to the edge of the surgical incision (Fig. 10.4) [4].

This technique is cheap, easy to apply, and immediately available [4]. However, it was abandoned after the 1980s because of multiple disadvantages: the risk to increase IAP at the closure, the impossibility to remove intra-abdominal fluids, and the impossibility to assess the intra-abdominal contents without reopening the abdomen [4, 8]. In trauma patients with increased IAP after an organ packing a zipper could be applied [4].

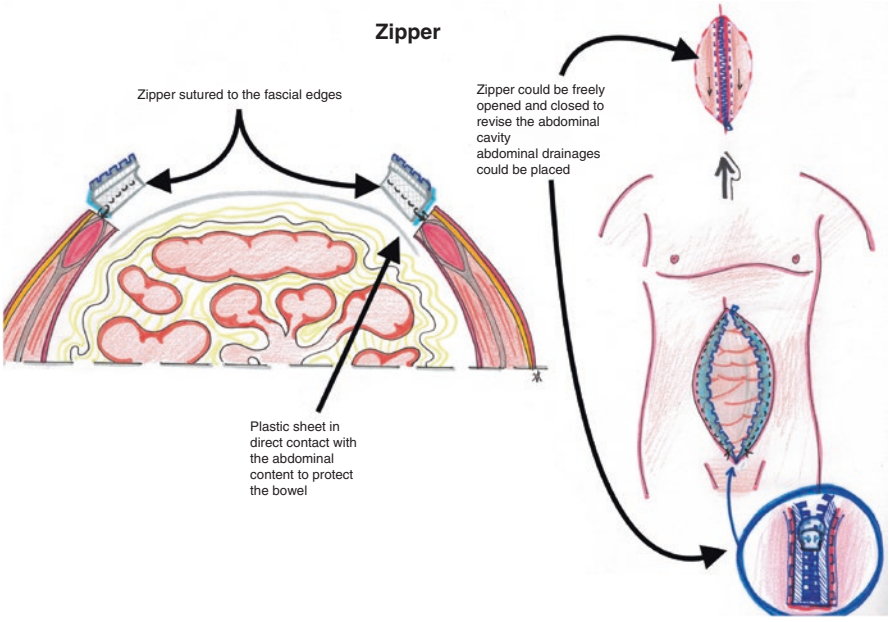

Zipper
Zipper sutured to the fascial edges
Zipper could be freely opened and closed to revise the abdominal cavity
abdominal drainages could be placed
Plastic sheet in direct contact with the abdominal content to protect the bowel

Fig. 10.4 Zipper (authorized reproduction from www.clinicalregisters.org)

10.2.5 Wittmann Patch

The Wittmann Patch (WP) is a TAC where two opposite Velcro sheets are sutured to the fascial edges. At the abdominal re-exploration, the two sheets are overlapped in the middle of the opening abdomen allowing a gradual re-approximation every 24–48 h of the two edges (Fig. 10.5) [2–4]. The WP is an easy technique and cheap and allows re-exploring easily the abdominal cavity. Recently a systematic review

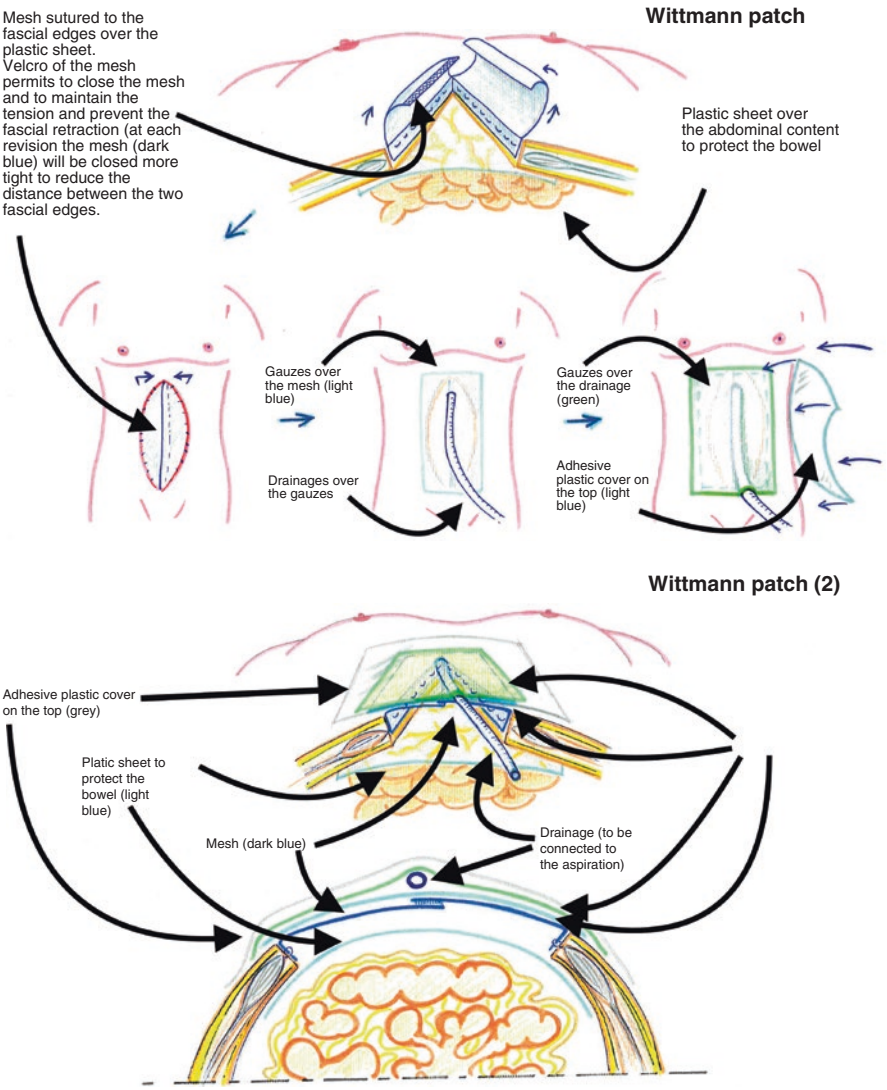

Wittmann patch
Mesh sutured to the fascial edges over the plastic sheet. Velcro of the mesh permits to close the mesh and to maintain the tension and prevent the fascial retraction (at each revision the mesh (dark blue) will be closed more tight to reduce the distance between the two fascial edges.
Plastic sheet over the abdominal content to protect the bowel
Gauzes over the mesh (light blue)
Drainages over the gauzes
Gauzes over the drainage (green)
Adhesive plastic cover on the top (light blue)
Wittmann patch (2)
Adhesive plastic cover on the top (grey)
Platic sheet to protect the bowel (light blue)
Mesh (dark blue)
Drainage (to be connected to the aspiration)

Fig. 10.5 Wittmann Patch (authorized reproduction from www.clinicalregisters.org)

reports a high rate of primary abdominal closure in patients treated with WP near to 77.8–94%, with a low mortality and complication rates (15.7% and 2.4–2.8%, respectively) particularly in non-septic patients [3, 5]. Literature reports no rates of incisional hernia in long-term follow-up with a low fistula rate of 0–4.2%, data that make this treatment safe and effective [5]. However, the disadvantage of WP is given from the impossibility to drain intraperitoneal fluid [4, 5].

10.3 Vacuum-Assisted Noncommercial Systems

In 2002 Miller et al. [11] revolutionized the open abdomen management substituting the surgical towels with a polyurethane sponge and attached a special drain to a dedicated pump for liquid aspiration (Fig. 10.6). A perforated plastic sheet covers the viscera; the sponge is placed above, between the fascial edges; the defect was covered by a Steri-Drape; and a suction drain connected to a pump is placed above the Steri-Drape [3]. The negative pressure created by the pump reduces intra-abdominal fluids, keeps a tension on the abdominal wall and the fascia, and removes intra-abdominal cytokines [12]. Since the 2000 many systems of negative pressure wound therapy (NPWT) are on the market (as Vacuum-Assisted Closure® (VAC),

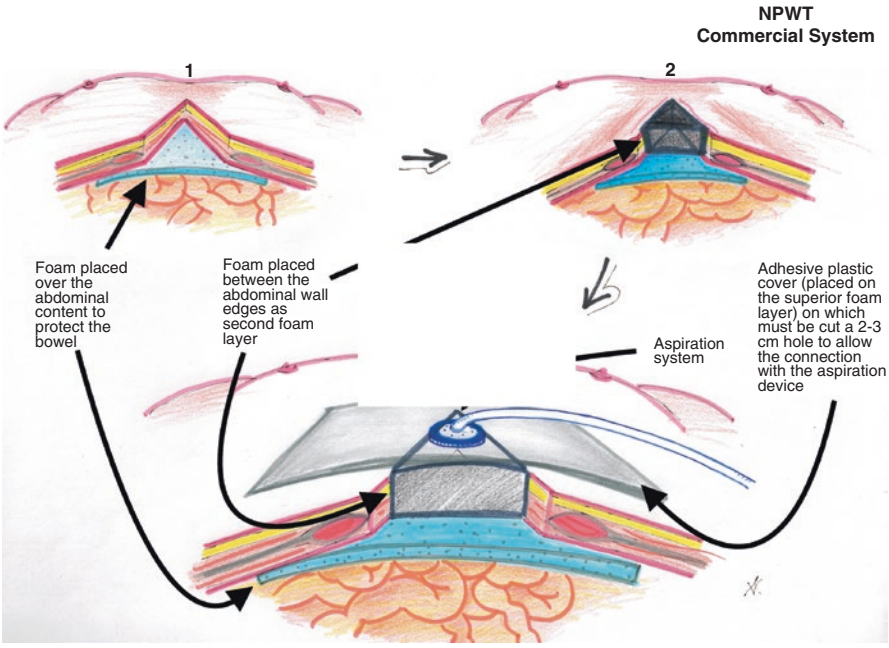

NPWT
Commercial System
1
2
Foam placed over the abdominal content to protect the bowel
Foam placed between the abdominal wall edges as second foam layer
Aspiration system
Adhesive plastic cover (placed on the superior foam layer) on which must be cut a 2-3 cm hole to allow the connection with the aspiration device

Fig. 10.6 VAC NPWT (authorized reproduction from www.clinicalregisters.org)

KCI, San Antonio, TX; ABThera™, KCI, San Antonio, TX; Renasis, Smith and Nephew, London, UK; CNP Suprasorb devices, Lohmann & Rauscher, Neuwied, DE; and others) [3, 4].

10.3.1 Barker Vacuum Pack

In 1995 Barker modified a negative pressure system described for the first time in South Africa by Schein in 1986 [2]. This technique was coined "Barker vacuum pack" (BVP) [13, 14]. The technique is a handmade negative pressure system and is realized putting a fenestrated, non-adherent sterile drape inside the abdomen to protect viscera, covered by two surgical towels or gauzes. Above the gauzes two large silicone drains like Jackson–Pratt drain are positioning and covering by other two gauzes, finally covering by a Steri-Drape over the wound to seal the abdominal cavity (Fig. 10.7) [2, 4]. The drains are connected to continuous wall suction at 100–150 mmHg [1, 5]. BVP is a successful technique with primary fascial closure rates from 35 to 92% with low fistula rates of 0–15% [5]. However, studying the distribution pattern of the present negative pressure of BVP has found a significant reduction of the pressure at the periphery [2]. This condition is minimized with modern devices [2].

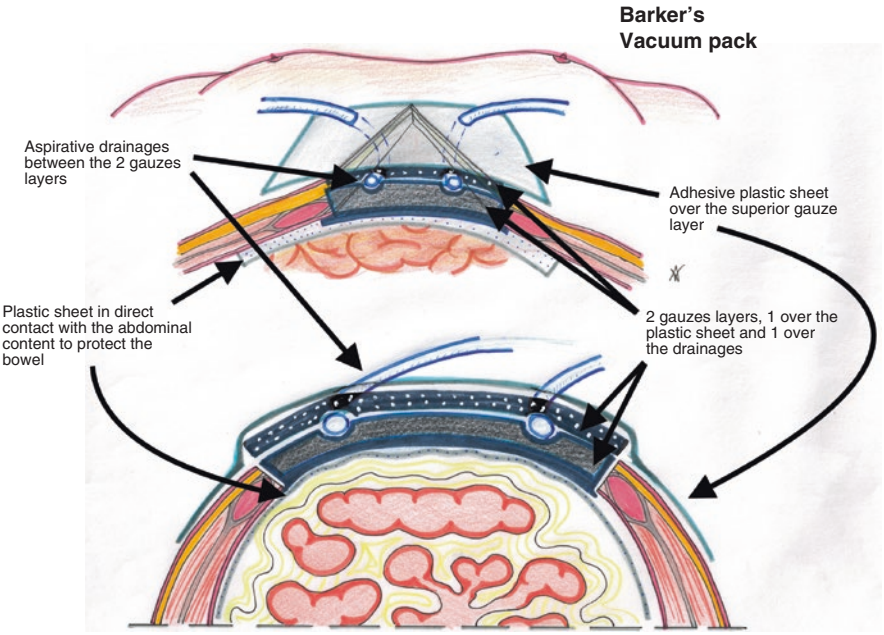

Barker's
Vacuum pack
Aspirative drainages between the 2 gauzes layers
Adhesive plastic sheet over the superior gauze layer
Plastic sheet in direct contact with the abdominal content to protect the bowel
2 gauzes layers, 1 over the plastic sheet and 1 over the drainages

Fig. 10.7 Barker vacuum pack (authorized reproduction from www.clinicalregisters.org)

10.4 Vacuum-Assisted Commercial Systems

10.4.1 Negative Pressure Wound Therapy (NPWT): ABThera (KCI), Renasis (Smith and Nephew), CNP Suprasorb Devices (Lohmann & Rauscher), and VivanoMed (Hartmann)

NPWT has become a common technique to manage open abdomen that includes older devices as VAC® to the new generation computerized pump as ABThera™ (2009) [1, 2, 4]. VAC® is a sophisticated NP dressing system that includes a polyurethane foam covered with a protected, fenestrated, non-adherent layer, connected with a tube to a canister and a computerized pump. This system avoids bowel–anterior abdominal wall adhesions and makes an abdominal re-exploration easy, putting the fascia in tension [2]. ABThera™ is a device composed of a visceral protective layer made of polyurethane foam with six radiating foam extensions enveloped in a polyethylene sheet with small fenestrations. This layer is placed into abdominal cavity under the abdominal wall to protect the bowel, into the paracolic gutters and pelvis. The characteristic of the intra-abdominal drape is to remove all the peritoneal fluid. The polyurethane foam is placed between the two incisional margins and is then covered with a sterile adhesive drape. A small piece of the adhesive drape and underlying sponge are excised, and an interface pad with a tubing system is applied over this defect and connected to a pump and a canister to collect fluids [2]. The dressing changes are usually performed every 48–72 h.

The advantages of the modern devices (of which ABThera™ is the prototype) comparing to the older (as Barker Vacuum Pack) are at first the possibility to applied an higher negative pressure from the periphery to the center of the system giving an improving efficacy to remove intra-abdominal toxin or bacteria-rich fluids and pro-inflammatory cytokines [1, 2, 4]. These devices are also easy for nursing and easy to change, reduce visceral edema, and maintain strength between the muscular edges [1, 2].

A recent systematic review reports better outcomes in terms of mortality in patients undergoing the VAC. In these cases mortality was lower than 25% (particularly in septic patients) [3]. However the authors report a fistulation rate of 15.6% in peritonitis and 7.3% in trauma patients [3]. Despite that VAC system and Wittmann Patch are considered (comparing to others open abdomen techniques). These are better systems (except for ABThera™ that was not included in this study) which allow to increase abdominal closure rates and reduce mortality and complications [3].

In a study by Cheathman [15], the 30-day primary fascial closure rate was neat to 70% in ABThera™ group and 51% in Berker group ($p = 0.03$), and also mortality is decreased in the first group ($p = 0.02$). In 2014 Kirkpatric et al. [16] report in the only randomized trial present in literature a lower 90-day mortality rate in ABThera group comparing to BVP group (HR, 0.32; 95% CI, 0.11–0.93; $p = 0.04$), with a similar incidence of primary fascial closure.

10.4.2 Tension Systems: Abdominal Re-approximation Anchor System (ABRA), NPWT Combined with Dynamic Fascial Suture, and Sequential Closure Technique

In the last years to increase the fascial closure rates, some re-approximation systems are applied. The problem of the abdominal closure is more present in patients undergoing OA for peritonitis, pancreatitis, vascular injuries, and ACS, compared to trauma patients, in which normally the closure is attained in the first 48–72 h [11, 17, 18]. One of them is the ABRA system (Canica, Almonte, Ontario, Canada). This system approximates the wound and the muscle edges through dynamic traction exerted by transfascial elastomers (Fig. 10.8) [19]. The elastomers (a series of midline-crossing elastic bands) are inserted during the surgical procedure through the full thickness of the abdominal wall, in a perpendicular manner at a distance of approximately 5 cm from the medial fascial margin, and then are aligned about 3 cm apart across the defect and fixed to the so-called button anchors at the insertion site. Also in that system, viscera are protected with a sterile fenestrated drape. To prevent the displacement and tilting, an adhesive button tail is attached to the

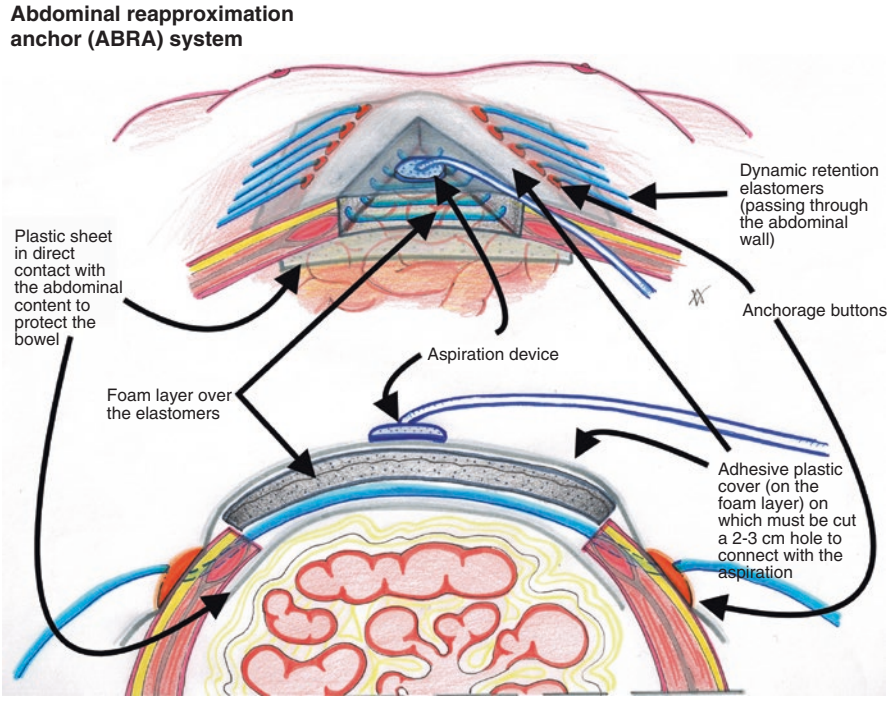

Abdominal reapproximation anchor (ABRA) system
Dynamic retention elastomers (passing through the abdominal wall)
Plastic sheet in direct contact with the abdominal content to protect the bowel
Anchorage buttons
Aspiration device
Foam layer over the elastomers
Adhesive plastic cover (on the foam layer) on which must be cut a 2-3 cm hole to connect with the aspiration

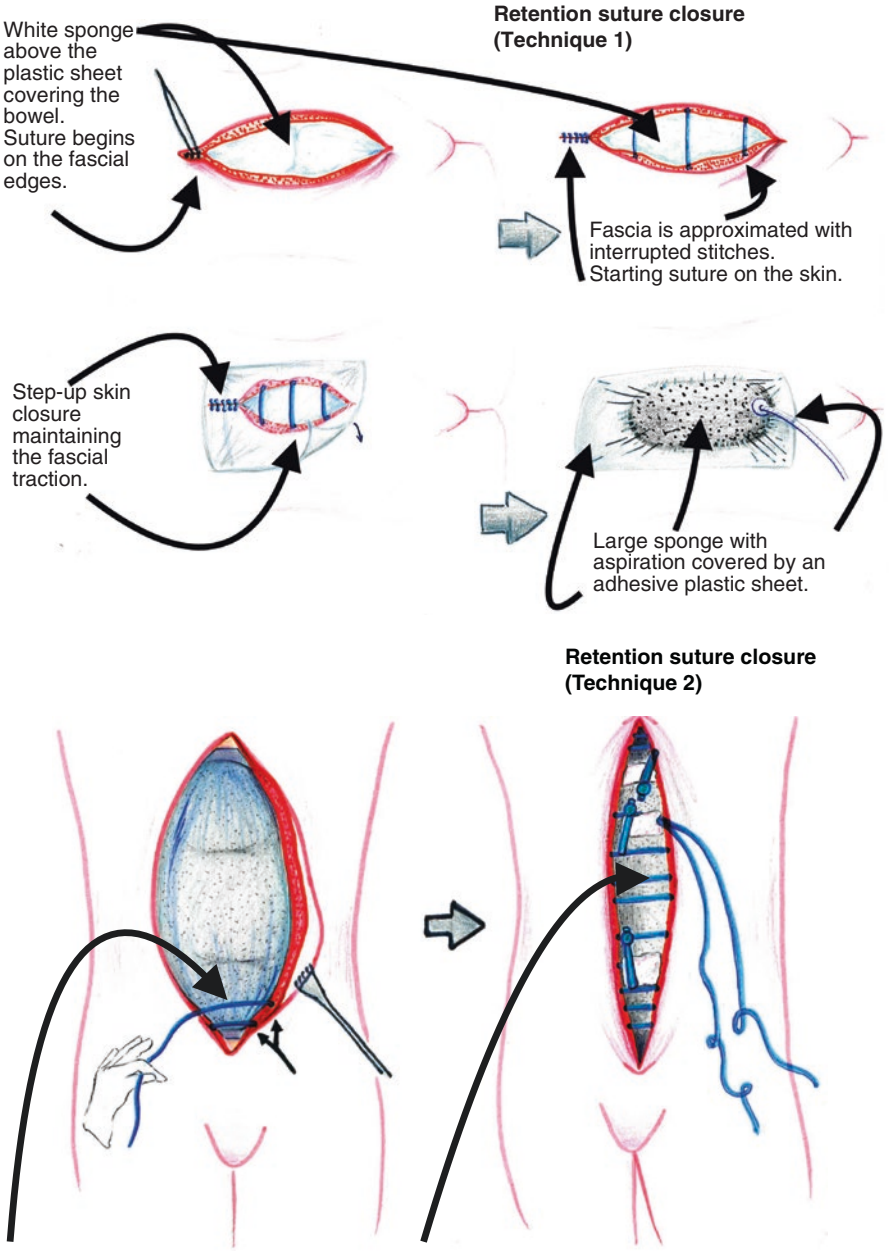
Fig. 10.8 ABRA (authorized reproduction from www.clinicalregisters.org)

anchor. A continuous dynamic traction is provided in a controlled manner between the elastomers and can be applied at the bed of patients avoiding a reintervention in the theater [20]. A NPWT can be applied in the defect during the margin re-approximation to drain intra-abdominal fluids [19]. When the fascial edges are near to 1 or less cm of distance, the surgical incision can be closed, the silicone sheet is removed, and the ABRA system can be removed. ABRA can achieve a delayed closure of the abdomen within 30 days (with a median duration of application of 7 days) in 61–88% of cases compared to 33–66% of closure rates in previous studies [19, 20]. However, that system can be painful, can be uncomfortable, and can give unsightly scarring and ulcerations [19]. Therefore, a reasonable tension should be applied.

The achievement of early closure is particularly difficult in patients who underwent OA for a peritonitis comparing to trauma patients. Atema in a systematic review [17] analyzing data from non-trauma patients reports an overall weighted rate of delayed fascial closure of 50.2%, with a range from 30 to 73.6% in patients treated with TAC techniques. In trauma patients, literature reports ranges of fascial approximation from 68 to 88% after 3.5 days to 9–9.9 days [21–23]. However, some authors like Burlew [24] suggest that also in those patients, a sequential closure technique performed by a systematic protocol would achieve a higher rate (100% in her series) of primary fascial closure. This technique consists in positioning multiple overlapping white sponges to cover the bowel, placing PDS sutures through the fascia, over the white sponges to prevent fascial retraction. A plastic adherent drape was placed to cover the white sponge. The central portion is removed by cutting along the wound edges, leaving only that adherent to the skin, and large black VAC sponges are placed on top of the white sponges and plastic-protected skin. The black sponges are affixed with an occlusive dressing, and standard suction with a commercial pump is applied [24]. During the relaparotomies the closure of the fascia is started from the lower or the upper of the surgical incision with an interrupted PDS suture, and in the center of the defect, re-approximation stitches are placed to prevent fascial retraction. Viscera are covered with white sponge and a NPWT is placed above the defect [24].

Other authors like Fortelny [25] suggest the same approach in non-trauma patients showing a fascial closure of 78.2% within 12.6 days. The technique was performed using a dynamic fascial suture using vessel loop with vertical stitches at a distance of 1.5–2 cm laterally to the incision with a subsequent re-approximation at every changing dress (Fig. 10.9) [25]. The enteroatmospheric fistula rate is 3.4% and the mortality rate is 55.2% [25].

Another sequential closure technique is the vacuum-assisted wound closure and mesh-mediated fascial closure (VAWCM) [5]. Acosta et al. [26] in a prospective multicentric study evaluate the fascial closure rates after VAWCM technique that results ranged from 78 to 89% with a fistula rate from 7 to 12%. This technique combined the use of a VAC system with the use of a mesh sutured to the fascial edges, the viscera under the mesh covered by a perforated polyethylene sheet, a


Retention suture closure
(Technique 1)
White sponge above the plastic sheet covering the bowel. Suture begins on the fascial edges.
Fascia is approximated with interrupted stitches. Starting suture on the skin.
Step-up skin closure maintaining the fascial traction.
Large sponge with aspiration covered by an adhesive plastic sheet.
Retention suture closure
(Technique 2)

Dynamic fascial suture using vessel loop in a continuous suture on the fascial edge (below a plastic sheet cover the bowel as the ABthera system, and above the suture a sponge covered by an adhesive plastic sheet and a suction pump).

Fig. 10.9 Retention interrupted suture and NPWT (figure in the *right*). Dynamic fascial suture using vessel loop with NPWT (figure in the *right*) (authorized reproduction from www.clinicalregisters.org)

sponge above the mesh, and a VAC system above the sponge [26]. During the changing dress, the mesh was cut and sutured in tension to reduce the space between the two edges. The advantages of this technique are to facilitate closure of the OA, by maintaining a clean abdomen and a tension on the fascial edges.

10.5 Open Abdomen Classification

In 2009 the World Society of the Abdominal Compartment Syndrome (WSACS) (2006–2007) has redefined the definition and the guidelines of the abdominal compartment syndrome performing a classification of the OA [27]. It was necessary to define a classification system to standardize clinical studies and to classify the pathology. Bjork and colleagues [27] proposed a classification, named Bjork's classification divided in four grades. Grade IA or IIA is clean with or without adherences between the bowel and abdominal wall, Grade IB or IIB is a contaminated abdomen with or without adherences between the bowel and abdominal wall, Grade IIIA is the OA complicated by a fistula formation, and the Grade IV is a frozen abdomen with bowel fixity unable to close surgically with or without fistula (Fig. 10.10).

In that year a new revision of that classification was performed to give a major comprehension of the complexity of the OA [28]. In the last classification, the major revision was done considering the different roles especially in terms of patient outcomes of the presence of the enteric leak compared to the enteroatmospheric fistula that is a more critical condition.

BJORK CLASSIFICATION 2009		BJORK CLASSIFICATI ON 2016	
GRADE	DESCRIPTION	GRADE	DESCRIPTION
1 A	Clean OA without adherence between bowel and abdominal wall or fixity	1 A	Clean OA without adherence between bowel and abdominal wall or fixity
B	Contaminated OA without adherence/fixity	B	Contaminated OA without adherence/fixity
		C	Enteric leak, no fixation
2 A	Clean OA developing adherence/fixity	2 A	Clean OA developing adherence/fixity
B	Contaminated OA developing adherence/fixity	B	Contaminated OA developing adherence/fixity
		C	Enteric leak, developing fixation
3 A	OA complicated by fistula formation	3 A	Clean, frozen abdomen
		B	Contaminated, frozen abdomen
4	Frozen OA with adherent/fixed bowel; unable to close surgically; with or without fistula	4	Established enteroatmospheric fistula, frozen abdomen
	fistula		frozen abdomen

Fig. 10.10 Comparison between the old and the new Bjork classification

Conclusions

Literature offers different techniques to manage an OA; however, none of these are still considered the ideal technique to achieve rapid closure without fascial short- and long-term complications. It is therefore surgeon's responsibility to know and to learn how to use these techniques, by adapting to the type of pathology and to the type of open abdomen to be treated.

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The Role of Instillation in Open Abdomen Management

11

Martin Rosenthal and Marc de Moya

Key Points

- Limited data to support direct peritoneal resuscitation (DPR).
- DPR has been shown in animal models to decrease need for intravenous crystalloid.
- DPR has been suggested to improve ability to perform delayed primary closure of open abdomen.

11.1 Introduction

The indications for open abdomen are unstable patients in shock due to trauma, abdominal sepsis, and severe acute pancreatitis and in general situations in which there is the potential for ongoing development of intra-abdominal hypertension (IAH), in order to prevent the development of abdominal compartment syndrome (ACS). Damage control surgery includes (1) controlling bleeding and contamination in the abdominal cavity and (2) leaving the abdomen open, to decompress, reevaluate, or facilitate return at planned relaparotomy. While damage control laparotomy (DCL) with the accompanied open abdomen has been shown to improve survival, this comes at a cost of a host of complications including fistulae, intra-abdominal infections, and the inability to perform fascial closure. Studies have shown that a delay greater than 7 days to fascial closure results in worse patient outcomes [1, 2]. Many strategies have been implemented to decrease these complications since the introduction of DCL

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including conservative intravenous fluid resuscitation strategies, hypertonic saline IV resuscitation, and temporary abdominal closure (TAC) including negative pressure wound therapy.

Despite improvements by using these adjuncts, DCL still suffers from a less than 100% fascial closure rate along with delays to successful fascial closure which leads to intra-abdominal infections, fistulae, and ventral hernias. A group from the University of Louisville has focused on studies using human and animal models of hemorrhagic shock with direct peritoneal resuscitation (DPR), whereby a hypertonic fluid is administered to the open abdomen in conjunction with negative pressure wound therapy to counteract the effects of shock. Their work has shown an increase in the rate of delayed primary fascial closure, a decreased time to fascial closure, as well as reduced intra-abdominal complications [2–4]. DPR appears to improve outcomes by splanchnic vasodilation reducing organ ischemia. This also effectively reduces organ edema as well as the pro-inflammatory cytokine cascade. In animal shock models, they were able to show a reduction in mortality from 40% to 0% [2, 3, 5]. Specific findings will be discussed below in regard to both animal and human studies.

11.2 Pathophysiology of Hemorrhagic Shock in Trauma

In addition to the effects of the open abdomen, i.e., lateral wall retraction, there are other physiologic factors that can lead to inability to close the abdomen and/or worsening inflammatory response. Trauma patients in hemorrhagic shock are often aggressively resuscitated with IV crystalloid fluid and blood products to maintain intravascular volume and restore normal hemodynamics. Unfortunately, measurements of blood pressure, heart rate, urine output, and central venous pressure used commonly as clinical endpoints of adequate resuscitation are inadequate indicators of tissue perfusion [6, 7]. Thus, conventional IV resuscitation from trauma and hemorrhagic shock sometimes culminates in multisystem organ failure, over-resuscitation, and delayed primary abdominal closure. This can be attributed to three major pathophysiologic events, progressive splanchnic vasoconstriction and hypoperfusion, gut-derived exaggerated systemic inflammatory response, and obligatory tissue fluid sequestration [3, 8, 9].

During shock the body experiences a profound vasoconstriction of both the pulmonary and systemic circulation. Even after normalization of hemodynamics, the vasoconstriction resolves slowly. The visceral organs such as the small intestine and the liver are particularly prone to prolonged ischemia. When these organs are reperfused, they create a severe and prolonged pro-inflammatory response along with damage to tight junctions between endothelial cells that promotes bacterial translocation and organ edema [10].

11.3 Direct Peritoneal Resuscitation

DPR involves bathing the abdominal contents with a dextrose-based, vasoactive, topical, hypertonic, dialysate solution (Delflex, Fresenius Medical Care). The technique is described by Zakaria, Garrison et al. in which after DCL the abdomen is prepared for temporary abdominal closure [3]. A 19Fr silicone drain is placed in the left upper lateral quadrant and directed around the root of the mesentery along the left paracolic gutter and down into the pelvis. A temporary abdominal closure is prepared with suction catheters tucked into towels superficial to a plastic sheet draped on the surface of the bowel, and an occlusive dressing is then applied (Fig. 11.1). The abdomen is than lavaged with Delflex, starting with a 800–1000 mL bolus through the left upper quadrant drain, followed by a continuous infusion of 400 mL/h until repeat laparotomy. The dialysate fluid is continuously suctioned through the superficial drains, and IV resuscitation is given concomitantly [3].

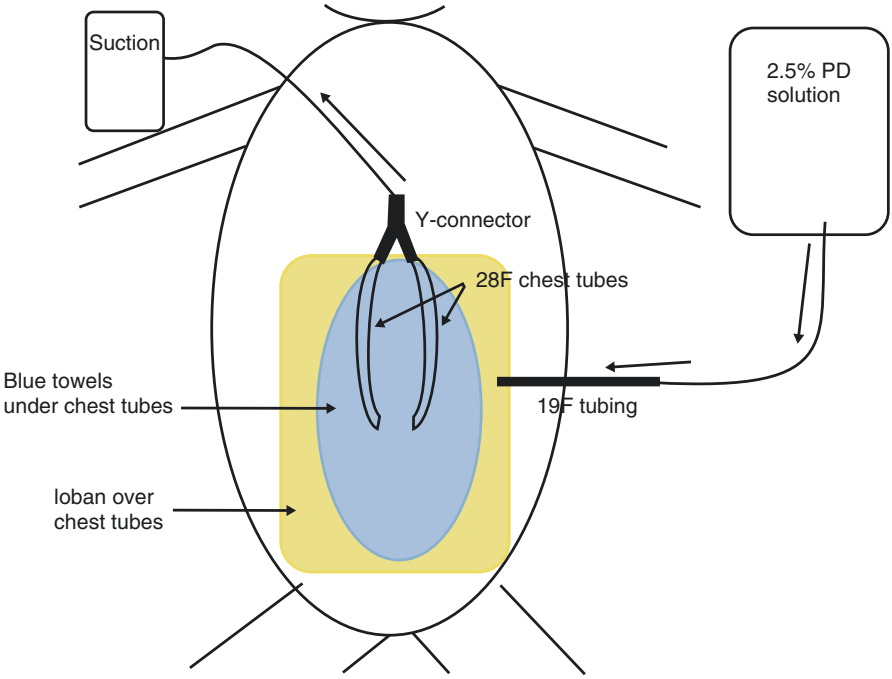

Fig. 11.1 Model of direct peritoneal resuscitation. Reprinted with permission from Weaver et al. [10]

11.3.1 Animal Studies

In previous microcirculatory studies performed by Zakaria and Garrison et al., peritoneal dialysis fluid was shown to preserve endothelial cell function, reverse established vasoconstriction, and restore intestinal blood flow above baseline [2, 6, 7, 9, 10]. This led to further studies on whole animals in a hemorrhagic shock model where rats were exposed to isotonic saline versus Delflex abdominal lavage after being bled to shock levels. They were able to demonstrate that the suffusion of a 2.5% glucose-based peritoneal dialysis solution (Delflex) concurrent with intravenous resuscitation from hemorrhagic shock causes microvascular vasodilation and increases visceral and hepatic blood flow, reverses endothelial cell dysfunction, improves survival and downregulates the inflammatory response, reverses established microvascular constriction, normalizes capillary perfusion density, and normalizes systemic water compartments [6]. In addition they noted a marked ability to decrease visceral edema and normalize body water ratios [5]. Delflex DPR leads to these physiologic changes without a systemic change in mean arterial pressure [9].

11.3.2 Human Studies

The first human trial was completed by Smith and Garrison et al. in 2010 [5]. They performed a retrospective study of 20 trauma patients undergoing DCL with Delflex DPR with 40 matched controls. They were able to demonstrate a significantly decreased time to definitive abdominal closure and an increased rate of abdominal closure with DPR (4.4 versus 7 days, p 0.003) [5]. The odds ratio for intra-abdominal complications after DCL was 5:1 in favor of those patients receiving DPR compared with controls (p 0.05). In addition, at 6 months the incisional hernia rate was significantly less than the matched controls. The DPR group required an equivalent volume of resuscitation as the matched controls without changes in the mean arterial pressure. However, their resuscitation involved over 20 L of fluid in the first 24 h [5]. Smith and Garrison et al. following their previous work performed a prospective study on DPR in 2014 including 88 patients with abdominal catastrophes including pancreatitis, perforated hollow viscous, bowel obstruction, and ischemic enterocolitis [3]. The DPR group had a significantly higher rate of fascial closure (43 versus 68%, p 0.02) and shorter length of time to definitive fascial closure (5.9 versus 7.7 days, p 0.03). They also demonstrated a lower APACHE II and sequential organ failure assessment (SOFA) score at 48 h and fewer abdominal complications than controls. The number of ventilator days and ICU length of stay were also significantly reduced in the DPR group. DPR led to less IV crystalloid resuscitative fluid compared to controls (18,300 mL versus 15,900 mL, $p < 0.001$) at 48 h [3] (Table 11.1). Of note, however, in this study patients were resuscitated with larger than normal volumes of crystalloid which has been shown to negatively impact patient outcomes [4, 11]. This may partly explain the benefits of DPR in this patient population and may not be applicable

Table 11.1 Outcomes from prospective human study of DCR

	Controls ($n = 44$)	DPR ($n = 44$)	p
No. of trips to operating room	4 (2)	3 (2)	0.02
Time to abdominal closure, d	7.7 (4.1)	5.9 (3.2)	0.02
Primary fascial closure, *n* (%)	19 (43)	29 (68)	0.03
No. abdominal complications	21 (47%)	12 (27%)	0.04
Ventilator days	14 (6)	10 (5)	0.01
ICU LOS, d	24 (11)	17 (9)	0.002
Total LOS, d	41 (13)	35 (16)	0.06
ICU-free days	26 (11)	31 (13)	0.05
Mortality, *n* (%)	12 (27)	7 (16)	0.15

Reprinted with permission from Smith et al. [3]

under new conservative resuscitation protocols. In addition, their rate of closure (70%) was far less than that seen in other studies using IV hypertonic saline, Wittmann Patch, or protocoled sequential fascial closure. The study also suffered from a surgeon bias due to a lack of adequate blinding.

Conclusion

DCL and the open abdomen, initially brought into the mainstream by Retondo et al. 1993, have shown improved outcomes in trauma patients and septic open abdomens. With the advent of this new technique came new complications associated with it. DPR was developed to counteract some of the components of the pathophysiology of shock and the large-volume resuscitations commonly encountered in its management with overall success. Animal models have elegantly demonstrated the benefits of DPR on endothelial cell dysfunction, reversal of splanchnic vasoconstriction, and decreased fluid sequestration all leading to a decreased systemic inflammatory response and reduced cellular hypoxia. Thus far only one prospective human trial has been published demonstrating increased time to fascial closure and overall increased rate of fascial closure compared to contemporary resuscitation controls with fewer abdominal complications. Other advances such as conservative IV fluid resuscitation, IV hypertonic saline, and temporary abdominal closure products at this time have more convincing data to support their benefit in the open abdomen, and further studies will be needed using DPR in conjunction with these strategies.

Take Home Messages

1. Limiting fluid resuscitation improves ability to close the open abdomen.
2. Direct peritoneal resuscitation lacks ample evidence to support its routine use.
3. DPR may reduce the need for intravenous crystalloid resuscitation.

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The Open Abdomen in Infants and Children

12

Davide Corbella, Oliviero Fochi, and Mirco Nacoti

Key Points

Despite the long-standing experience in staged closure and open abdomen treatment, there is a wide spread reluctance to implement these treatments in the pediatric pediatric patients. Whereas the open abdomen has a sound and recognized role in the adult literature, the pediatric one lags behind showing that:

- Intra-abdominal pressure is not measured on a routine basis.
- Cut-off of intra-abdominal hypertension is often not standardized in the clinical practice.
- The definition of abdominal compartment syndrome appears to be not so well understood in the pediatric intensivist community.
- Open abdomen treatment is often reserved for "hopeless" cases with poor results.

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12.1 Introduction

The deleterious effect of an elevation in the intra-abdominal pressure (IAP) has been described in the nineteenth century, but relevant research on this topic did not happen until the 1980s. Intra-abdominal hypertension (IAH) and acute compartment syndrome (ACS) were first defined by the World Society of the Abdominal Compartment Syndrome (WSACS) in 2006. This society was the first to undertake a systematic review of the literature and to produce evidence-based recommendations [1] as well as the first set of guidelines for diagnosis and management [2]. In 2013 they produced the first specific pediatric recommendations and definitions [3]. Despite the recent interest, IAH and ACS have long been familiar concepts when dealing with patients with abdominal wall defects. Staged abdominal wall closure and open abdomen treatment were part of the routine practice of neonatal surgery since the first work of Gross [4] in the late 1940s, which showed a better outcome when the abdomen was closed without pressure in a staged manner.

With the exception of those neonatal and transplant cases, the deleterious effect of an IAH and the subsequent ACS is still quite low as demonstrated by two surveys taken in the pediatric community. Kimball [5] in 2006 in the USA showed that a quarter of pediatric intensivists were unaware of how to measure a bladder pressure, with one third of them that would have never performed a decompressive laparotomy. In 2012 Kaussen's study [6] found that nearly 80% of German pediatric intensivists relied only on clinical signs to pose the diagnosis of ACS.

The low confidence with this topic is reflected, and influenced, by the low amount of specific pediatric data. Moreover the general quality is low with a total lack of multicentric prospective studies or implementation of national registries.

12.2 Pathophysiology of ACS and Pediatric Considerations

The common final pathway of cellular damage of every compartment syndrome is cellular death subsequent to ischemia. The arteriovenous pressure gradient theory is the most widely accepted pathophysiologic mechanism to explain the hit during compartment syndrome [7]. This theory assumes that tissue perfusion is equal to the gradient between mean arterial pressure (MAP) and mean venous pressure. When the intra-compartment pressure exceeds the venous pressure, it becomes the limiting factor to blood flow. The decrease of end-organ perfusion leads to ischemia and subsequently swelling, capillary leak, cellular edema, and more intra-compartment pressure that eventually ends in cellular death.

Compliance and perfusion pressure are the main variables of this model. Pediatric patients are peculiar from this point of view as compliance of the compartment is different from adults and perfusion pressure (i.e., MAP) is generally lower and a function of age.

12.2.1 Policompartment Syndrome

What has become clear in the last 10 years is the fact that an increase in pressure in any of the closed compartments of the body leads to an increase in the other compartment (i.e., central nervous system, thoracoabdominal, pulmonary, ocular, limbs). This led to the definition of polycompartment syndrome [8, 9]. This syndrome and the possible treatment with decompressive laparotomy and decompressive craniotomy have been described by Scalea [10] in 2007 when he treated 102 trauma patients with multiple compartment decompression. Interestingly the decompressive laparotomy brought to a decrease in the ICP as a direct evidence of the close relationship between the two compartments. The abdominal compartment plays a cornerstone role being upstream to the lower limb and downstream to the thorax. This explains the predominant cardiovascular effect when this compartment is affected. The interrelationship between the compartments is synthetized by Fig. 12.1. The figure briefly

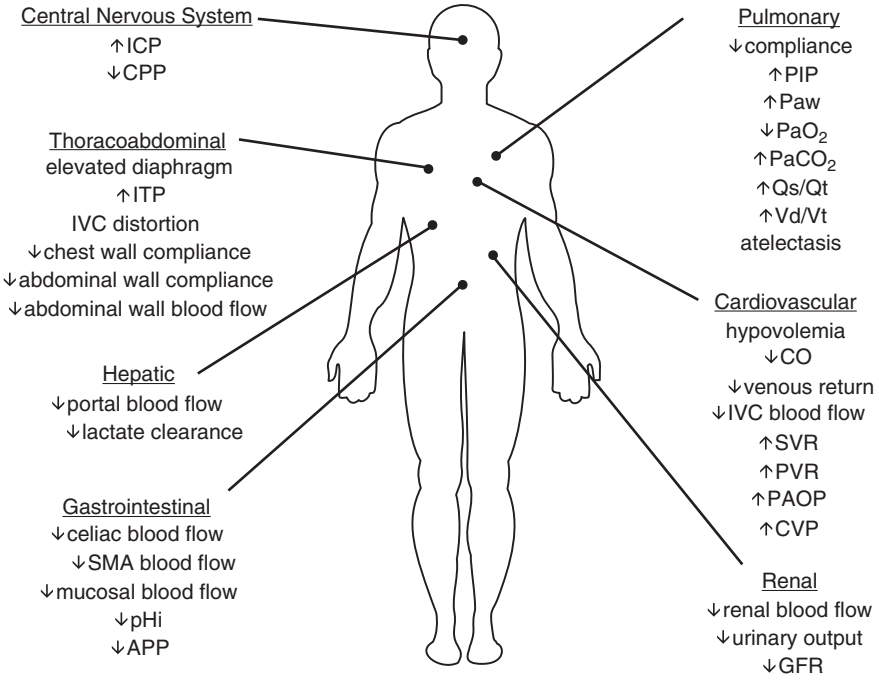

Fig. 12.1 Pathophysiologic implications of intra-abdominal hypertension. The effects of intra-abdominal hypertension are not limited just to the intra-abdominal organs, but rather have an impact either directly or indirectly on every organ system in the body. *ICP* intracranial pressure, *CPP* cerebral perfusion pressure, *ITP* intrathoracic pressure, *IVC* inferior vena cava, *SMA* superior mesenteric artery, *pHi* gastric intramucosal pH, *APP* abdominal perfusion pressure, *PIP* peak inspiratory pressure, *Paw* mean airway pressure, *PaO2* oxygen tension, *PaCO2* carbon dioxide tension, *Qs/Qt* intrapulmonary shunt, *Vd/Vt* pulmonary dead space, *CO* cardiac output, *SVR* systemic vascular resistance, *PVR* pulmonary vascular resistance, *PAOP* pulmonary artery occlusion pressure, *CVP* central venous pressure, *GFR* glomerular filtration rate. (Reproduced with permission from [11], © Cheatham; licensee BioMed Central Ltd. 2009)

presents the main physiologic derangements associated with an increase in pressure for every compartment and explains how this leads to a decrease of end-organ perfusion in the others [11].

Pediatric data on this regard are scanty and often of low quality. However, the pathophysiologic model developed in the adult population and in the animal studies is sound. We are legitimated to translate it in the pediatric cohort of patients keeping in mind some distinctive features as the differences in compartment compliance and perfusion pressure.

12.3 Definition of Abdominal Compartment Syndrome

ACS was historically defined as organ failure associated to an increase in abdominal compartment pressure (measured or supposed by clinical evaluation) that is reversed or dramatically improved by open abdomen treatment [12]. We report in Table 12.1 the differences between adult and pediatric patients from the WASC consensus conference [3].

12.4 Measurement of Intra-abdominal Pressure and IAP Cutoffs

Clinical estimation of IAP is unreliable as pointed out by Sugrue [13] in the adult population. Direct measurement of the IAP by a peritoneal catheter is just unfeasible, with the relevant exceptions of the post-cardiovascular surgery patient [14] or patients with an abdominal drain in place (if it is kept reasonably patent). The intravesical pressure is the actual measurement of choice [2]. Once abdominal contraction is ruled out by sedation or neuromuscular block, the volume of instillation inside the bladder is the main source of bias. The correct volume of instillation in the adult population has been investigated by several papers as a volume too great

Table 12.1 Main differences between adult and pediatric patients according to the WSACS definitions

Pediatric definitions	Adult definitions
ACS in children is defined as a sustained elevation in IAP of greater than 10 mmHg associated with new or worsening organ dysfunction that can be attributed to elevated IAP	ACS is defined as a sustained IAP > 20 mmHg (with or without an APP > 60 mmHg) that is associated with new organ dysfunction/failure
The reference standard for intermittent IAP measurement in children is via the bladder using 1 mL/kg as an instillation volume, with a minimal instillation volume of 3 mL and a maximum installation volume of 25 mL of sterile saline	The reference standard for intermittent IAP measurements is via the bladder with a maximal instillation volume of 25 mL of sterile saline
IAP in critically ill children is approximately 4–10 mmHg	IAP is approximately 5–7 mmHg in critically ill adults
IAH in children is defined by a sustained or repeated pathological elevation in IAP > 10 mmHg	IAH is defined by a sustained or repeated pathological elevation in IAP > 12 mmHg

can lead to a false high and a too low volume to a false low IAP reading [15, 16]. Davis [14] compared several indirect IAP measurements against the direct measurement of IAP by a peritoneal catheter in 20 kids admitted to a PICU after cardiac surgery with a peritoneal dialysis catheter. The indirect methods to estimate IAP were an intragastric manometer and an intravesical pressure at different volumes of normal saline. They found that the most accurate way to estimate the IAP was via a bladder catheter when a 1 mL/kg infusion of normal saline was used. Eijke [16] estimated the best volume to infuse in the bladder by an analysis of the bladder compliance curve with an increasing volume of normal saline. This study collected data from 96 pediatric patients admitted to a medical–surgical PICU on mechanical ventilation and without signs, symptoms, or risk factors of ACS. Considering the bladder compliance as a sigmoid curve, they defined the optimal volume as the one used to reach the lower inflection point. They confirmed that an infusion of 1 mL/kg of normal saline was the most accurate.

Devices to measure the IAP in the pediatric ICU are usually custom-made (see Fig. 12.2 for the system in use in our PICU).

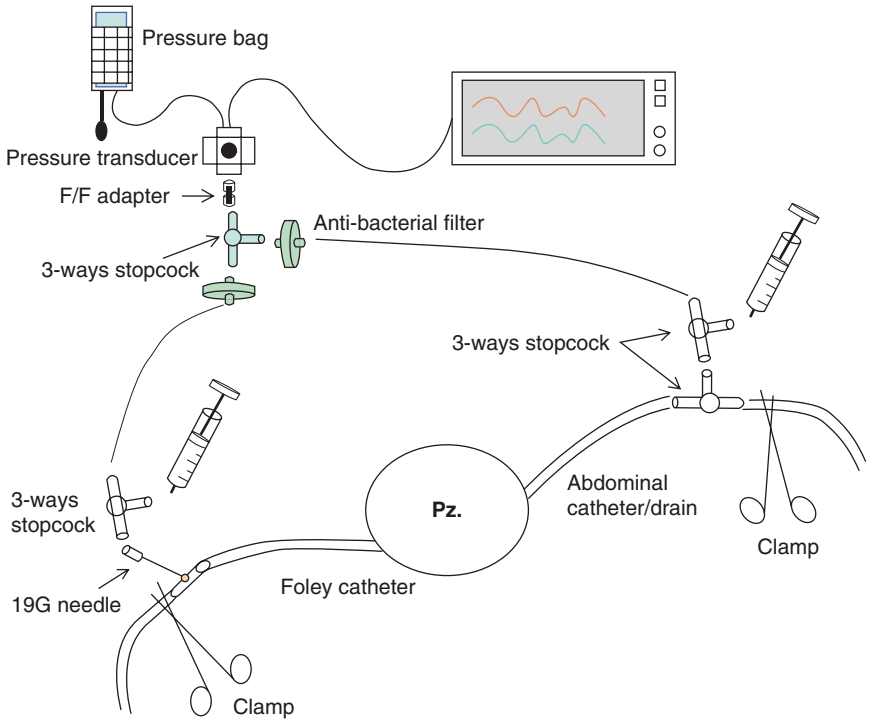

Fig. 12.2 Custom-made IAP measurement system (left side via a Foley catheter while on the right via an abdominal catheter drain). This system is essentially made by a regular urinary catheter, a three-way stopcock with attached on one end to the Foley catheter and on the others to a pressure transducer zeroed at midaxillary line and an infusion bag. After emptying the bladder and ensuring that there's no contraction of the abdominal wall muscles, an infusion of sterile normal saline into the bladder is performed according to the WSACS guidelines (1 mL/kg with a range from 3 to 25 mL)

IAP cutoffs to define IAH are lower in pediatric patients than in adults (10 instead of 20 mmHg). Different authors showed that organ failure is dramatically improved by decompressive laparotomy (DL) with a lower baseline IAP. Beck [12] in 2001 reported a 5-year retrospective study in which ten patients were treated with DL. Interestingly they had ACS with an IAP as low as 16 mmHg. The incidence of ACS was low (0.6%) reflecting the conservative threshold for intervention. Mortality was high (60%) and the authors suggested a too delayed timing for DL. Pearson [17] in 2010 published a cohort of 26 emergent DLs in the presence of ACS. ACS was defined as new-onset organ dysfunction in the presence of an IAP > 12 mmHg. They had a 58% mortality and advocated for earlier intervention as every patient dramatically improved after DL. More recently Rollins [18] showed how DL performed with an IAP > 20 mmHg in patients on extracorporeal membrane oxygenation (ECMO) yielded a 100% mortality. A threshold of 10 mmHg has been chosen as a cutoff point for the definition of IAH. This cutoff takes into account the normal values of IAP in critically ill kids (7 ± 3 mmHg) [16] and the evidence of an increase in mortality and ACS with IAP as low as 12 mmHg.

Indications and contraindications to IAP measurements are similar to adults and reported elsewhere.

12.5 Epidemiology and Outcome

Epidemiology of ACS and IAH in the pediatric population is unknown. The lack of awareness of this syndrome in the pediatric intensivist community has been already discussed [5, 6]. No national or society registry has been implemented until now. Moreover custom-made definitions of IAH and ACS were quite common, especially when defining the number of organ failure or the IAP cutoff (e.g., 20 instead of 10 [18]). All published case series are monocentric and show an incidence of ACS between 0.6 and 9.9% with mortality as high as 100% for ECMO patients (see Table 12.2). The only nationwide report is from Turkey, where Horoz [19] performed a 1-day national inquiry in 11 PICUs. Four IAP measurements were performed at 6 h intervals to all pediatric patients admitted in that day. Patients had to be admitted for treatment and for a period longer than 24 h with no contraindication to IAP measurement. The total sample was 130 patients, 46% of which had IAH. The IAH group had higher lactate levels and higher incidence of hypothermia and mechanical ventilation. Even though patients with IAH had a similar hospital and PICU length of stay and mechanical ventilation days to the non-IAH, they had a slightly higher mortality and higher rate of organ failure. The study couldn't rule out if the IAH was just a proxy of criticality or had any causal effect in the development of organ failure and death. Surprisingly when using a cutoff at 10 mmHg, almost half of the population is in the IAH group, greatly affecting the test specificity.

Table 12.2 List of studies exploring incidence and mortality of IAP\ACS in pediatric patients, according to their definitions

First author (year)	Type of study	Definition of ACS/IAH	Incidence	Mortality in patients with ACS
Beck (2001) [12]	Single center, prospective observational	IAP > 15 mmHg + two between oliguria/anuria, respiratory decompensation, hypotension/shock, metabolic acidosis	10 patients out of 1762 (0.6%)	60%
Divarci (2016) [36]	Single center, prospective observational	WASCS definition	IAH 9% ACS 4%	16%
Ejike (2007) [37]	Single center, prospective observational	IAP > 12 + one new organ dysfunction	14/294 (4.7%)	50%
Fontana (2014) [25]	Single center, retrospective, all pediatric kidney transplant	IAP > 20 + one new organ dysfunction	9/420 (2.1%)	None reported
Gupte (2010) [26]	Single center, retrospective, all pediatric intestinal transplant	Not defined	4/49	50%
Horoz (2015) [19]	Nationwide 1-day survey on the day of admission	WSACS definition	IAH 60/130 (46.2%)	46.7% (30-day mortality)
Pearson (2010) [17]	Single center, retrospective, patients undergoing emergent laparotomy	IAP > 12 + onset of new organ failure	26/264 (9.9%)	58%
Steinau (2011) [38]	Single center, retrospective	IAP > 12 + onset of new organ failure	Unknown	6/28 (21.4%)
Thabet (2016) [39]	Single center, prospective observational	WSACS definition	IAH (22/175) 12.6% ACS (7/175) 4%	6/7 (85.7%)
Neville (2000) [40]	Single center, retrospective	Abdominal distension and respiratory and/or cardiac failure	Unknown	35%

12.6 Special, and Critical, Clinical Scenarios

12.6.1 Abdominal Wall Defects

Gastroschisis, omphalocele, and Cantrell syndrome are congenital defects of the abdominal wall [20]. In 1948 Gross [4] proposed a staged closure of the abdomen, and since then the concept of "abdominal domain" or better to "regain abdominal

domain" has been mainstream in neonatal surgery in order to avoid the detrimental effect of ACS. Rizzo [21] proposed in 1996 the intraoperative vesical pressure to guide wall closure. However, only recently, Schmidt [22] showed that the use of intraoperative IAP via a bladder catheter could substantiate and standardize the choice on how to close the abdominal wall. In their prospective study, a cutoff of IAP > 20 mmHg was chosen for a staged closure. They had no difference between the staged and primary closure groups in terms of frequency of complications, time to begin oral feeding, and length of parenteral nutrition or hospital stay.

12.6.2 Congenital Diaphragmatic Hernia (CDH)

ACS develops in CDH patients when the organs herniated in the thorax are returned into the abdomen in the presence of a mismatch between the volume of those organs and the abdominal domain. Incidence of ACS and IAH are unknown in CDH patients. Data from the Canadian network of pediatric surgeons [23] reported an ACS incidence <1% and a delayed abdominal closure, based on clinical judgment, of 10%. This study suggests an actual or perceived risk of ACS in at least 10% of patients. A better and "justified" definition of an IAH to attempt a primary closure should be object of study as the open abdomen treatment in these patients is related with a prolonged hospitalization, morbidity, and ventilator days. Moreover the sudden development of ACS can have detrimental effect in an already unstable patient, as the sudden increase in IAP can compromise cardiac output. The mismatch between organs and domain may explain the observation of Dotta [24] that showed a decrease in cerebral NIRS during CDH repair, with the lowest values registered when the organs are placed in the abdominal cavity.

12.6.3 Solid Organ Transplantation

Solid organ transplantation with an adult graft is frequently complicated by some degrees of ACS due to a mismatch between abdominal domain and adult graft. The ideas at the basis of the use of a staged abdominal closure are to gain space by allowing graft or bowel edema to reabsorb or to wait for the abdominal cavity to stretch around the organs with a second-intention closure without compromising graft perfusion. This has been described for every abdominal or retroperitoneal organ. Fontana [25] reported nine cases of ACS out of 420 kidney transplants in his 25-year experience. All of them were pediatric patients who received an adult graft. This brought his group to measure IAP during abdominal closure on a routine basis. Intestinal or combined intestinal–liver transplantation with a mismatch between recipient and donor is a well-known, although rare, cause of ACS. Gupte [26] reports that since 2005 staged closure of the abdomen and pre-transplant abdominal tissue expanders were applied routinely whenever a mismatch between donor and

recipient was found. This is the result of their previous experience of ACS with 50% mortality. Sheth [27] reported similar results in the cohorts of patients from Necker. Delayed primary closure of the abdomen in pediatric liver transplantation was considered an emergency therapy when important bowel edema, massive transfusion, great donor graft mismatch, or bowel distension were detected [28]. Some centers now report an incidence of delayed closure of 50%. This shows a passage from an emergency treatment to a standard procedure when a risk of developing ACS is foreseen [29].

12.6.4 Necrotizing Enterocolitis

Necrotizing enterocolitis (NEC) is characterized by extensive damage to the bowel ranging from edema to necrosis and perforation. ACS is a well-known feature of the NEC [30]. Anyway it not clear whether it has a causal role in the development of the NEC or it just worsens the prognosis when the vicious circle of ischemia more pressure more ischemia is started. Staged closure of the abdominal cavity was reported as routinely performed by 25% of European pediatric surgeons [31] if a "tense closure" was suspected. In a study from Tanriverdi [32], an increase of IAP pressure between serial measurements was defined as an early sign of NEC. Interestingly, important abdominal impairment was detected for pressures as low as 10 mmHg [30, 32], questioning the value of 20 mmHg that is currently felt as a safe threshold for closing the abdomen in other neonatal diseases (i.e., wall defects [22]).

12.6.5 Pediatric Cardiac and ECMO Patients

Children requiring surgery for congenital heart disease have a number of perioperative risk factors for gut mucosal injury: young age, abnormal circulatory physiology (duct-dependent circulation and single ventricle), suboptimal mucosal perfusion, altered blood flow and hypothermia induced during cardiopulmonary bypass, and surgical trauma. These factors may predispose to splanchnic hypoperfusion, disruption of the gut's barrier function with development of endotoxemia, colonic distension, and IAH [33, 34].

Patients on ECMO are a peculiar challenge. An increased IAP reduces the return to the venous cannula impairing perfusion, but an open abdomen treatment is burdened by the risk of an unmanageable hemorrhage in anticoagulated patients. To date three papers report the experience of ACS in pediatric patients on ECMO. Rollins [18] in his cohort of seven patients that underwent laparotomy had no survival, whereas Prodhan [35] had two survivors out of four patients treated with peritoneal catheters. No formal IAP measurement was implemented. However, Rollins suggests possibly better results had decompressive laparotomies been performed earlier.

Take-Home Message

- IAP should be performed routinely in every critically ill pediatric patient.
- An IAP > 10 mmHg should be considered an "ominous" sign and prompt a more intensive monitoring of the patients even in those scenarios where a different cutoff has been used before.
- DL should be considered early in the development of organ dysfunction and not be used as the "last hope for hopeless cases."
- When possible those cases should be reported in literature, and more efforts should be poured in collecting data in prospective registry or trials.

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Intensive Care Unit Management of the Adult Open Abdomen

13

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Key Points

- Serial intra-abdominal pressure (IAP) measurements are essential in the critically ill patient at risk for intra-abdominal hypertension (IAH) and/or abdominal compartment syndrome (ACS).
- Multimodality medical management is effective at reducing elevated IAP when implemented early.
- Prompt surgical decompression should be performed in patients whose elevated IAP is refractory to nonoperative management strategies.

13.1 Introduction

Elevated intra-abdominal pressure (IAP) is a common pathophysiologic finding among critically ill patients [1]. Unfortunately, clinical recognition of this disease by critical care physicians and nurses remains low [2, 3]. As a result, it is frequently overlooked as a cause for patient deterioration until significant organ injury has occurred, resulting in patient morbidity, increased resource utilization, and unnecessary mortality. The causative factors for pathologic increases in IAP are as diverse as the patient populations that are at risk. Sepsis, traumatic injury, abdominal infection, chronic kidney or liver dysfunction/failure, ileus, pancreatitis, and burns, among others, have all been implicated in the development of intra-abdominal hypertension (IAH) and abdominal compartment syndrome (ACS). Traditionally considered a disease of the surgical patient, IAH/ACS may well be more common among medical patients where its development and presentation is typically more insidious [4].

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In this chapter, we will describe a comprehensive, evidence-based approach to the management of the ICU patient at risk for elevated IAP. This strategy has been developed over the past 20 years during the treatment of thousands of both medical and surgical patients with IAH/ACS and has been demonstrated to significantly reduce patient morbidity and mortality [5].

13.2 Pathophysiology

To understand the efficacy of the therapeutic tactics advocated in this strategy, an understanding of the pathophysiologic implications of elevated IAP is essential. IAP is determined by three factors: [1] abdominal organ volume, [2] presence of space-occupying substances (such as blood, ascites/fluid, air, or tumor), and [3] abdominal wall compliance. Therapies implemented to reduce the injurious effects of elevated IAP must therefore confront one of these three factors. Normal adult IAP is less than 5 mmHg, but IAP in the post-laparotomy patient is typically 10–15 mmHg. In the critically ill patient with septic shock, an IAP of 15–20 mmHg is common. Patients with systemic hypoperfusion and organ dysfunction/failure commonly demonstrate an IAP of 20–30 mmHg or greater. These pressures can have a catastrophic impact upon organ perfusion and function leading to the significant morbidity and mortality associated with both IAH and ACS. Although abdominal decompression significantly improves survival in such patients, contrary to popular belief, IAP does not become zero once a patient's abdomen is open. IAH and ACS may both occur despite the presence of an open abdomen and temporary abdominal closure.

IAP, however, is only part of the equation. As a result of patient variability, there is no single-threshold IAP value that can be globally applied to the decision-making of all patients. IAP alone lacks sufficient sensitivity and specificity at the clinically appropriate thresholds of 10–25 mmHg to be useful as a resuscitation endpoint. Abdominal perfusion pressure (APP), calculated as mean arterial pressure (MAP) minus IAP, assesses not only the severity of IAP present, but also the adequacy of the patient's systemic and visceral perfusion. APP has been demonstrated to be superior to both IAP and global resuscitation endpoints, such as arterial pH, base deficit, and arterial lactate, in its ability to predict patient outcome [6]. It represents an easily calculated parameter for guiding the resuscitation and management of the patient with IAH/ACS, having been demonstrated to exceed the clinical prediction of IAP alone in several clinical trials.

Clinical examination through abdominal palpation has a sensitivity of less than 50% for determining the presence of elevated IAP. Therefore, if IAH is suspected to be present, IAP must be measured. Failure to identify IAH and/or ACS when present is associated with reported mortality rates of up to 100%. When recognized and appropriately treated, mortality can still reach 30–40% depending upon the etiology of the disease process [5]. Serial determinations of IAP have been shown to reliably detect the development of IAH and facilitate early treatment of ACS, with significant reductions in patient morbidity and mortality. This is especially true in the patient with an open abdomen where IAP and APP become essential resuscitative

parameters. Regrettably, studies demonstrate that many physicians and nurses do not understand how to measure IAP or are reluctant to measure IAP in their patients at risk [2, 3].

Elevated IAP causes significant impairment of cardiac, pulmonary, renal, gastrointestinal, hepatic, central nervous system, and abdominal wall perfusion and function, with each organ demonstrating its own unique vulnerability. This differential response to IAP, coupled with the augmented susceptibility seen in the presence of hypovolemia and comorbid disease, further complicates the management of these complex patients. The detrimental effects of IAP on each of these organ systems are described in Table 13.1. The possibility of IAH

Table 13.1 Pathophysiological implications of IAH/ACS

Organ system	Pathophysiological effects	Clinical manifestations	Threshold IAP
Cardiovascular	Decreased preload/venous return Increased afterload Compression of inferior vena cava	Decreased cardiac output Increased susceptibility to hypovolemia	10 mmHg
Pulmonary	Increased intrathoracic pressure Cephalad elevation of diaphragm Extrinsic compression of pulmonary parenchyma Alveolar atelectasis Increased airway resistance	Hypoxemia Hypercarbia Elevated airway pressures Increased intrapulmonary shunt Increased alveolar dead space	15 mmHg
Renal	Decreased renal blood flow Renal vein compression Renal parenchymal compression	Oliguria Anuria Acute renal failure	15 mmHg
Gastrointestinal	Decreased mesenteric blood flow Intestinal ischemia Bacterial translocation/ sepsis	Increased susceptibility to hypovolemia Increased visceral edema/ capillary leak Metabolic acidosis	10 mmHg
Hepatic	Decreased hepatic vein blood flow Decreased portal vein blood flow	Hepatic dysfunction/ failure Metabolic acidosis	10 mmHg
Central nervous system	Increased intrathoracic pressure Decreased cerebral venous outflow	Increased intracranial pressure Decreased cerebral perfusion pressure	15 mmHg
Abdominal wall	Decreased abdominal wall compliance Decreased rectus sheath blood flow	Fascial dehiscence	10 mmHg

IAP intra-abdominal pressure, *IAH* intra-abdominal hypertension, *ACS* abdominal compartment syndrome

should be considered in any patient who presents with one or more of the following: prolonged shock (acidosis, hypothermia, hemorrhage, coagulopathy), visceral ischemia/perforation, traumatic injury, sepsis, massive fluid resuscitation (>5 L in 24 h), ruptured abdominal aneurysm, retroperitoneal hemorrhage, abdominal neoplasm, liver dysfunction/ascites, pancreatitis, burns, or ileus/gastroparesis.

Finally, the severity of IAP is less important than the duration of IAH. Prolonged elevations in IAP result in organ dysfunction and failure that can have a significant impact upon patient morbidity and mortality [7]. Every effort should be made to reduce the period of time that a critically ill patient's IAP exceeds 15 mmHg, the threshold at which most IAP-induced organ dysfunction occurs. The duration of IAH and/or development of ACS correlate significantly with increased ICU and hospital length of stay, patient care costs, duration of mechanical ventilation, and patient mortality [8, 9].

13.3 Intensive Care Unit Management

While surgical decompression is widely and erroneously considered the only treatment for IAH/ACS, nonoperative medical management plays a vital role in both the prevention and treatment of IAP-induced organ dysfunction and failure [10] (Fig. 13.1). Appropriate management of IAH/ACS is based upon four general principles:

1. Serial monitoring of IAP
2. Optimization of systemic perfusion and end-organ function
3. Institution of organ-specific therapies to reduce IAP and avoid the detrimental end-organ consequences of IAH/ACS
4. Prompt surgical decompression for refractory IAH/ACS

13.3.1 Sedation and Analgesia

Pain, agitation, ventilator dyssynchrony, and use of accessory muscles during work of breathing may all lead to increased thoracoabdominal muscle tone and decreased abdominal wall compliance, resulting in elevated IAP. Appropriate patient sedation and analgesia can reduce muscle tone and potentially decrease IAP to less detrimental levels. In addition to ensuring patient comfort, therefore, adequate sedation and analgesia also serve a useful therapeutic role in the patient with IAH. The goal should be to reduce IAP to less detrimental levels and raise APP above 60 mmHg to ensure adequate systemic perfusion. In patients with significant elevations in IAP, sedation and analgesia to a level of general anesthesia may be necessary to overcome increased abdominal wall tone.

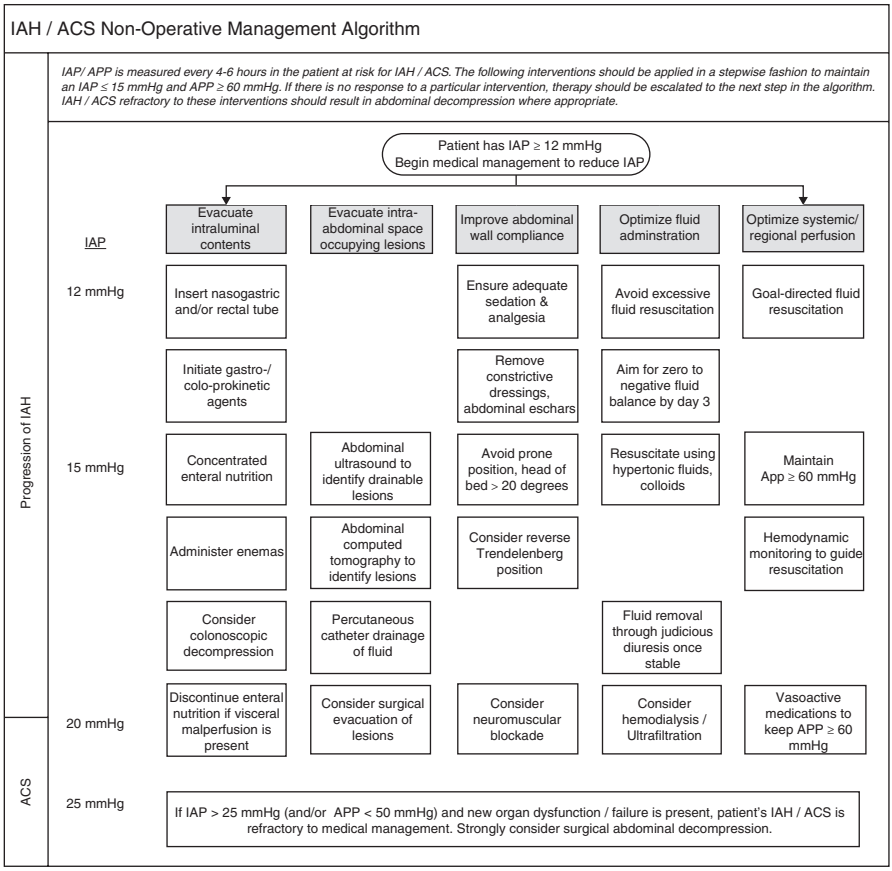
IAH / ACS Non-Operative Management Algorithm
IAP/ APP is measured every 4-6 hours in the patient at risk for IAH / ACS. The following interventions should be applied in a stepwise fashion to maintain an IAP ≤ 15 mmHg and APP ≥ 60 mmHg. If there is no response to a particular intervention, therapy should be escalated to the next step in the algorithm. IAH / ACS refractory to these interventions should result in abdominal decompression where appropriate.
Patient has IAP ≥ 12 mmHg
Begin medical management to reduce IAP
Progression of IAH
ACS
IAP
Evacuate intraluminal contents
Evacuate intra-abdominal space occupying lesions
Improve abdominal wall compliance
Optimize fluid adminstration
Optimize systemic/ regional perfusion
12 mmHg
Insert nasogastric and/or rectal tube
Ensure adequate sedation & analgesia
Avoid excessive fluid resuscitation
Goal-directed fluid resuscitation
Initiate gastro-/ colo-prokinetic agents
Remove constrictive dressings, abdominal eschars
Aim for zero to negative fluid balance by day 3
15 mmHg
Concentrated enteral nutrition
Abdominal ultrasound to identify drainable lesions
Avoid prone position, head of bed > 20 degrees
Resuscitate using hypertonic fluids, colloids
Maintain App ≥ 60 mmHg
Administer enemas
Abdominal computed tomography to identify lesions
Consider reverse Trendelenberg position
Hemodynamic monitoring to guide resuscitation
Consider colonoscopic decompression
Percutaneous catheter drainage of fluid
Fluid removal through judicious diuresis once stable
20 mmHg
Discontinue enteral nutrition if visceral malperfusion is present
Consider surgical evacuation of lesions
Consider neuromuscular blockade
Consider hemodialysis / Ultrafiltration
Vasoactive medications to keep APP ≥ 60 mmHg
25 mmHg
If IAP > 25 mmHg (and/or APP < 50 mmHg) and new organ dysfunction / failure is present, patient's IAH / ACS is refractory to medical management. Strongly consider surgical abdominal decompression.

Fig. 13.1 Intensive care unit management of IAH/ACS. *IAP* intra-abdominal pressure, *APP* abdominal perfusion pressure, *IAH* intra-abdominal hypertension, *ACS* abdominal compartment syndrome. Modified with permission from: Cheatham ML, World J Surg. 2009;33:1116–1122

13.3.2 Nasogastric/Colonic Decompression, Prokinetic Motility Agents

Gastrointestinal ileus is common among patients who have had abdominal surgery, peritonitis, major trauma, significant fluid resuscitation, or electrolyte abnormalities, many of which are independent risk factors for IAH/ACS. Excessive air and fluid within the hollow viscera, as a space-occupying structure, can raise IAP and lead to organ dysfunction and failure. Nasogastric and/or rectal drainage, enemas, and even endoscopic decompression are relatively noninvasive methods for reducing IAP and treating mild to moderate IAH in patients with visceral distention. Administration of prokinetic motility agents such as erythromycin,

metoclopramide, or neostigmine is also useful in evacuating intraluminal contents and decreasing visceral volume. All patients with elevated IAP should undergo nasogastric decompression (with colonic decompression if clinically indicated). This simple and commonly overlooked maneuver can frequently reduce IAP, raise APP, improve visceral perfusion, and decrease the need for more aggressive interventions.

13.3.3 Patient Positioning/Avoidance of Constrictive Dressings

Appropriate patient positioning can significantly impact IAP. The classic Fowler's patient position with both head and feet elevated compresses the abdominal cavity between both the rigid ribcage and pelvis, resulting in elevated IAP. Maintaining the spine and legs in the same axis avoids this unnecessary abdominal compression and can reduce IAP and improve APP. When head of bed elevation is necessary to improve respiratory effort, minimize pulmonary aspiration, or facilitate treatment of traumatic brain injury, use of the reverse Trendelenburg position can accomplish all of these goals simultaneously while avoiding abdominal compression and elevated IAP [11]. Abdominal binders and constrictive dressings should be avoided for similar reasons as these can also increase IAP. In burn patients, abdominal escharotomy is particularly effective in reducing IAP and improving APP.

13.3.4 Goal-Directed Fluid Resuscitation

Hypovolemia aggravates the pathophysiologic effects of elevated IAP, while hypervolemia (i.e., excessive crystalloid volume resuscitation) is an independent predictor for the development of ACS. The fluid status of patients at risk for IAH/ACS should be carefully scrutinized to avoid over-resuscitation. Careful monitoring and maintenance of urinary output at no more than 0.5 mL/kg/h is appropriate. Fluid losses from an open abdomen, if present, must be considered for accurate patient fluid balance assessment. High-rate maintenance fluid infusions should be avoided as this tends to result in excessive fluid administration over time. When necessary, frequent, small-volume as opposed to large-volume fluid boluses should be utilized to avoid over-resuscitation. Hypertonic crystalloid and colloid-based resuscitation have been demonstrated to reduce IAP and decrease the risk of iatrogenic, resuscitation-induced, increases in IAP. In critically ill patients, invasive hemodynamic monitoring using volumetric-based monitoring technologies can be very useful in assessing intravascular volume status and optimizing patient resuscitation. Traditional pressure-based parameters such as pulmonary artery occlusion pressure and central venous pressure have been found to be inaccurate in the presence of elevated intra-abdominal and intrathoracic pressure and can lead to erroneous clinical decisions regarding fluid status.

13.3.5 Diuretics and Continuous Venovenous Hemofiltration/ Ultrafiltration

Early intermittent hemodialysis or continuous hemofiltration/ultrafiltration may be more appropriate than continuing to volume load the patient and increase the likelihood of secondary ACS with its attendant morbidity and mortality. Fluid output from an open abdomen actually serves as a form of peritoneal dialysis and can help avoid the development of acute renal failure in the anuric/oliguric patient. Diuretic therapy, in combination with colloid, may be considered to mobilize third-space edema and reduce IAP once the patient is hemodynamically stable. These therapies must be utilized with caution, however, as they tend to decrease APP and may worsen the patient's systemic perfusion if not carefully monitored.

13.3.6 Neuromuscular Blockade (NMB)

Diminished abdominal wall compliance due to pain, tight abdominal closures, and third-space fluid can increase IAP to potentially detrimental levels. NMB has been reported to be an effective method for reducing IAP in early IAH. A brief trial of NMB for 24–48 h can be useful, in conjunction with other interventions, to reduce IAP and allow resolution of the patient's IAH, thus avoiding the need for decompressive laparotomy. NMB is not efficacious in the presence of advanced IAH or ACS, where delays in decompression will only serve to worsen the patient's end-organ failure. The potential benefits of NMB therapy must be balanced against the risks of prolonged paralysis.

13.3.7 Mechanical Ventilation

As described in Table 13.1, elevated IAP causes cephalad deviation of the diaphragm, resulting in increased airway pressures and compression of the pulmonary parenchyma. As a result, such patients are at risk of acute respiratory failure and the need for prolonged mechanical ventilatory support. The majority of such patients are appropriately managed using traditional volume-based modes of ventilation. Patients are optimally ventilated using 6–8 mL/kg ideal body weight (not actual body weight). Pressure-limited modes of ventilation are useful in patients with significant elevations in peak and plateau airway pressures, recognizing that IAP raises baseline intrathoracic pressure necessitating reevaluation of the therapeutic goals typically used in patients without IAH. Positive end-expiratory pressure (PEEP) is commonly necessary to maintain alveolar volumes and combat cephalad elevation of the diaphragm due to IAP. At the moment of abdominal decompression, however, the physician or respiratory therapist must be prepared to immediately reduce the level of PEEP administered as the now unopposed excursion of the diaphragm caudally can result in barotrauma to the lungs. A general rule of thumb is to reduce the

pre-decompression level of PEEP by 50% and then titrate the patient's PEEP based upon their subsequent oxygenation.

Patients who require abdominal decompression and maintenance of a temporary open abdomen commonly require mechanical ventilation postoperatively. Traditionally, such patients have been left intubated throughout the duration of their open abdomen. Recent evidence, however, demonstrates that such patients can be successfully extubated prior to definitive abdominal closure (Sujka et al., unpublished data). This significantly reduces the total duration of mechanical ventilation, decreases the risk for ventilator-associated pneumonia and need for tracheostomy, and reduces both ICU and hospital length of stay. Predictors of successful extubation include higher Glasgow Coma Scores and lower Injury Severity Scores (especially the Chest Abbreviated Injury Score component) suggesting that patients who are more alert, able to participate in post-extubation pulmonary rehabilitation, and less severely injured are good candidates for early extubation despite an open abdomen.

13.3.8 Nutritional Support

Appropriate and timely nutritional support is crucial to the successful resuscitation and management of any critically ill patient. Early enteral nutrition, once the patient's acute shock state has been corrected and adequate visceral perfusion is present, helps to prevent the development of ileus and bacterial translocation and improves wound healing. Parenteral nutrition, due to its infectious complications and increased cost, should be reserved for those patients who develop a high-volume enterocutaneous fistula or intestinal malabsorption. Patients with mild to moderate IAH can be successfully fed enterally, even with an open abdomen. In fact, enteral nutrition helps to reduce intestinal edema and can speed the process of definitive abdominal closure. Enteral nutrition should be held in the presence of low APP due to concern for worsening visceral ischemia. Nutritional support should begin with a caloric goal of 30 kcal/kg/day and protein goal of 1.5 gm/kg/day. High-protein, high-calorie formulas allow nutritional requirements to be met with decreased total feeding volumes and less potential for worsening the patient's IAP. Our preference is to use 2 kcal/mL formulas as the total daily feeding volume is generally well tolerated even in the presence of moderate IAH. It is important to account for additional protein losses from the open abdomen, if present, by replacing each liter of peritoneal fluid lost with 12.5 gm of protein (2 gm of nitrogen) [12].

13.3.9 Vasoactive Medications

In order to maintain an adequate APP in the presence of elevated IAP, the patient's blood pressure (and therefore MAP) may need to be supported using vasoactive medications such as norepinephrine. This therapy should be implemented only after ensuring adequate intravascular volume administration to avoid causing unnecessary

vasoconstriction and worsening visceral ischemia. The use of vasoactive medications to augment APP in the euvolemic patient, however, helps to avoid excessive volume resuscitation and is more easily titrated than fluid administration.

13.3.10 Percutaneous Decompression

Percutaneous catheter drainage of free intra-abdominal fluid, air, abscess, or blood is an effective technique for reducing IAP and potentially correcting IAH-induced organ dysfunction. Performed under ultrasound or computed tomography guidance, percutaneous decompression can significantly reduce IAP and decrease the need for and morbidity of surgical decompression. This minimally invasive approach to IAH/ACS management is most effective in patients with secondary ACS due to excessive resuscitation, burns, acute pancreatitis, or ascites [13]. Patients with IAH/ACS refractory to percutaneous catheter decompression should undergo urgent abdominal decompression.

13.3.11 Abdominal Decompression

Surgical decompression of the abdomen has long been the standard treatment for IAH/ACS [5, 14, 15]. It can be lifesaving when a patient's organ dysfunction and/or failure are refractory to medical treatment. Delayed abdominal decompression and disregard of high IAP levels are associated with significant increases in patient mortality. Prophylactic decompression and creation of a temporary abdominal closure in surgical patients at risk for elevated IAP significantly reduce the subsequent development of IAH/ACS and improve survival. Emergent decompression may be performed either in the operating room or at the patient's bedside in the intensive care unit if cardiopulmonary instability precludes safe transport. While seemingly aggressive and disabling, patients at risk for IAH/ACS who are treated with abdominal decompression demonstrate identical long-term physical and mental health function as well as resumption of gainful employment compared to similar patients who do not require an open abdomen [8, 9]. This potentially lifesaving technique, therefore, should not be withheld from a patient who is demonstrating signs of ACS.

13.4 Strategy Overview

Based upon both clinical evidence and over two decades of high-volume experience, we share our management algorithm for the intensive care unit patient with elevated IAP. First, serial IAP measurements are performed liberally due to the significant incidence of IAH in the high-risk patient and its significant associated morbidity and mortality. Second, immediate abdominal decompression is performed in any patient who is found to have evidence of ACS. This procedure is appropriate given that early decompression significantly improves survival and the patient's open abdomen can generally be closed within the first week without

significant long-term residual physical or mental health deficits. Third, in the patient with IAH, but no ACS, APP is maintained above 60 mmHg through the implementation of the nonoperative interventions described above. We do not excessively resuscitate patients simply to maintain an APP above 60 mmHg if their organ perfusion and function are adequate with an APP between 50 and 60 mmHg. Fourth, inability to maintain a minimum APP of 50 mmHg is an indication for decompressive laparotomy and maintenance of an open abdomen, using a temporary abdominal closure until the patient's clinical status improves. Fifth, post-decompression monitoring of IAP continues as, contrary to popular belief, IAH and ACS can recur and visceral perfusion can still be inadequate despite an open abdomen. Inability to maintain an appropriate APP is an indication to decompress the abdomen further through either a larger laparotomy or placement of a more compliant temporary abdominal closure. Sixth, attempts to close the patient's abdomen after decompression are guided by the patient's IAP and APP. While same-admission primary fascial closure should always be the goal following decompressive laparotomy, persistent elevations in IAP with marginal APP calculations should lead to a surgical decision for either split-thickness skin grafting of the exposed viscera or skin-only closure, as opposed to attempts to tightly close the abdominal wall. Active communication between intensivist and surgeon is vital in the successful management of these patients. Inappropriate fascial closure commonly results in recurrent ACS, decreased visceral perfusion, and a high mortality rate.

Conclusion

The ICU patient with elevated IAP represents one of the most complex patients that an intensivist can care for. Such patients are at high risk for multisystem organ dysfunction and failure, especially if the patient's elevated IAP is not recognized and appropriately addressed in a timely fashion. The management strategy outlined above has been continuously developed in our ICUs during the care of thousands of patients with elevated IAP over the past two decades. It has been demonstrated to reduce the morbidity of IAH/ACS and significantly improve patient survival. Adoption of such a multimodality, evidence-based management strategy can be expected to achieve similar results in any ICU.

Take-Home Messages

1. IAP should be measured in all critically ill patients with evidence of organ dysfunction and/or failure.
2. Implemented early, multimodality nonoperative management can reduce IAP and prevent progression of IAH to ACS.
3. Immediate surgical decompression should be performed in the presence of ACS or when adequate end-organ perfusion and function cannot be maintained.
4. Patient survival from IAH/ACS is significantly improved by adopting a comprehensive ICU management strategy.

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Management of the Open Abdomen in PICU

14

Torsten Kaussen

14.1 Historical Development and Background

From a historical perspective, the open abdomen (OA; syn. abdomen apertum, laparostoma, temporary abdominal closure [TAC]) is a form of treatment in pediatric surgery that emerged with the development of methods to temporarily expand as well as reconstruct the abdominal wall within the framework of operative care for inborn abdominal wall defects [1–3]. It is in the context of abdominal wall approximation and adaptation that gastroschisis and omphalocele are still considered prototypes for diseases often leading to a predisposition for intra-abdominal hypertension (IAH) and abdominal compartment syndrome (ACS) [4–6].

One speaks of an IAH in children starting at an intra-abdominal pressure (IAP) of 10 mmHg [7]. According to Kron et al. [8], the pediatric gold standard for IAP quantification is the modified measurement of bladder pressure. Further methods are used—albeit much less often—in everyday clinical practice [9–14]. The most notable technique involves continual indirect measurement of IAP via the stomach (IAP-monitor of Fa. Spiegelberg®, Hamburg). Following increases in IAP of only 6–8 mmHg in premature infants and newborns, cardiorespiratory limitations have been observed and described [15, 16]; however, the most recent data does not allow for a definition of limits adapted to percentiles. Depending on the level of IAP values, four non-percentile-adjusted IAH severity grades apply (see Table 14.1) [17]. Starting at Grade II, there is an increased transition into a complete ACS defined by the additional occurrence or aggravation of pending organ dysfunction (OD; OD criteria, Table 14.2) [18].

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Table 14.1 Child-oriented adapted WSACS consensus definitions [7, 17]

IAP	Pressure within the abdominal cavity (mmHg, measured at end expiration)
Normal IAP	7 ± 3 mmHg in critically ill children
APP RFG IAH	=MAP−IAP (abdominal perfusion pressure = mean arterial pressure minus IAP) =MAP−2 × IAP (renal filtration gradient) Sustained or repeated pathological elevation in IAP ≥ 10 mmHg
– Grade I – Grade II – Grade III – Grade IV	IAP 10–12 mmHg IAP 13–15 mmHg IAP 16–18 mmHg IAP > 18 mmHg
ACS	Sustained IAP ≥ 10 mmHg associated with new organ dysfunction/failure
Primary ACS	Condition associated with injury or disease in the abdominopelvic region
Secondary ACS	Condition that does not originate from the abdominopelvic region
Recurrent ACS	Condition in which ACS redevelops after previous surgical or medical treatment of primary or secondary ACS

Table 14.2 Diagnostic criteria for organ dysfunction

Cardiovascular	Despite intravenous application of ≥40 mL/kg isotonic volume in 60 min persisting • Hypotension with BP <5th percentile for age or systolic BP < 2 SD below normal for age • Vasoactive drug therapy to keep BP in normal range • Two of the following – Arterial lactate >2 times upper limit of normal – Prolonged capillary refill >5 s – Oliguria, urine output <0.5 mL/kg/h – Metabolic acidosis (base deficit >5 mmol/L) – Core to peripheral body temperature difference >3°C
Hematologic	• Thrombocyte count <80,000/mm^3 or decline of 50% in thrombocyte count from the highest value recorded over the past 3 days • INR > 2
Hepatic	• Total bilirubin ≥4 mg/dL (not applicable for newborn) • ALT two times upper limit of normal age
Neurologic	• Glasgow Coma Scale (GCS) ≤11 • Acute change in mental status with decrease in GCS ≥3 points from abnormal baseline
Renal	Serum creatinine ≥2 times upper limit of normal for age Twofold rise in baseline serum creatinine
Respiratory	• Oxygenation index <300 in the absence of cyanotic heart disease or preexisting lung disease • $PaCO_2$ > 65 mmHg or increase of >20 mmHg over baseline • Proven need or FiO_2 > 0.5 in order to maintain saturation ≥92% • Need for mechanical ventilation (invasive or noninvasive)

Cardiovascular, hematologic, hepatic, neurologic, renal, and respiratory dysfunction according to the international pediatric sepsis consensus conference [18]
BP blood pressure, *GCS* Glasgow Coma Scale, *ALT* alanine aminotransferase

The various types of ACS are described in Table 14.1 [17]. Whereas primary ACS etiologies dominate in newborns and infants (e.g., abdominal wall hernia, necrotizing enterocolitis [NEC], meconium ileus with/without perforation, volvulus, invagination), secondary ACS forms occur more often in older children following systemic inflammatory processes with fluid diapedesis and capillary leakage [19] (e.g., sepsis/systemic inflammatory response syndrome [SIRS], burn, trauma, mass transfusion, overhydration, extracorporeal circulation).

The highest ACS prevalence lies at 20% and is found in neonatal intensive care unit (NICU) patients [17, 20]. This rate decreases as the patients age, lying at ~4% in adolescents [17, 21, 22]. In high-risk pediatric populations (e.g., abdominal wall closure, following organ transplantation (Tx), mechanical ventilation, and extracorporeal circulation), IAH incidence is even up to 80% [23–26], and ACS lies at 18–37% [22, 23, 27].

The steps taken for IAH and ACS prevention and therapy in children and adolescents do not differ from those taken in adults (Table 14.3) [7, 17]. Should conservative and interventional methods not achieve a quick and/or lasting reduction in IAP, the chosen responses are prompt operative decompression and, where necessary, the creation of a laparostoma [28]. Regarding urgency, the following surgical saying also applies to IAH and ACS: "The sun should not set and rise between diagnosis and final therapy." Although relief via an operation in cases of a pronounced IAP dynamic or incipient ACS can be decisive for survival, there is often a fatal delay before adequate therapy is initiated. According to a retrospective investigation in adults, a decompressive laparotomy occurs on average 18 h following diagnosis

Table 14.3 Therapeutic options to lower IAH [7, 17]

	Evacuation of intraluminal contents	Evacuation of intra-abdominal space occupying lesions	Improvement of abdominal wall compliance	Optimization of fluid administration	Optimization of abdominal (APP) and systemic perfusion
Medical, noninvasive options	Gastric/rectal tube diet		Analgetics and sedatives	Modest fluid administration	Goal-directed fluid administration
	Prokinetics		Positioning	Diuretics	Pressors/ Inotropes
	Fasting		Muscle relaxants		
Interventional, minimal-invasive options	Gastric/ colonoscopic decompression	Paracentesis		Continuous	
		Percutaneous catheter drainage		Venovenous hemofiltration	
Surgical, invasive options		Decompressive laparotomy	Escharotomy/ fasciotomy		Laparostomy ("TAC," temporary abdominal closure)

[29]. A comparable investigation in pediatric patients does not exist. Generally speaking, the indication for invasive methods in pediatrics is clearly more conservative. This is fatal insofar as persistence in immunological activation could be proven in animal models following an 18 h exposure to IAH using interleukin and TNF-α-indications (own unpublished data) [30–33].

Experiences as well as developments in and around the care of congenital abdominal wall and diaphragmatic hernias have helped pave the way to the different laparostomata used in today's pediatric surgery [3]. Given the discrepancy between the abdominal space available and volume required for the transfer of prolapsed organs in neonatal patients, therapeutic procedures enabling successful abdominal wall closures and the survival of most children affected were not possible until the 1940s [34]. This was after the development of the "Schuster procedure" and comparable ways of expanding the abdominal wall [35]. In addition to the incremental development of different surgical methods (e.g., only closing the skin, the Bogota pouch, Wittmann Patch, component separation, zipper, vacuum pack, VAC® therapy) and optimization via material used for plastic surgery (e.g., Silastic, absorbable and nonabsorbable artificial mesh, auto-/iso-/homo- and heterologous biomaterials) [36–46], progress in intensive care monitoring and therapy has especially contributed to the improvement in survival rates [47]. The type of laparostoma used is less important for intensive care. Besides modern cardiocirculatory and respiratory intensive management, the availability of parenteral nutrition [48, 49] and calculable anti-infective strategies can be viewed as evolutionary breakthroughs [50].

In the second half of the twentieth century, laparostoma therapy began to be applied for other indications. A typical procedure used in connection with abdominal sepsis and damage control surgery (with and without IAH/ACS), abdomen apertum, plays an important role in guaranteeing success in pediatric organ transplantation (Tx) [51] (above all in liver and multiorgan Tx but less often in kidney Tx). Even with the advent of split-liver Txs in 1988 and living organ donations in 1989 (which increased the availability of smaller transplants), as well as the ability to approximate the supply needed for infants [52], the volume of small split-liver transplants frequently exceeds the capacity of children's abdomens (large-for-size Tx) and requires temporary expansion of the abdominal wall followed by an incremental closure over a period of days or weeks [17, 53–55]. During this time the abdominal wall and cavity's capacity can expand and be adjusted where necessary.

14.2 Definition and Differentiation

In contrast to figures documented in the treatment of adults, those for prophylactically laid laparostomata in pediatric medicine are disproportionately higher than those for therapeutically indicated TACs. Various prophylactic OA forms are being established for neonatal and infant patients in the context of abdominal wall and diaphragmatic hernias as well as organ Txs. As patient age, level of physical activity, and personal mobility increase, the spectrum becomes more similar to that of adults, thus necessitating therapeutic laparostomata (e.g., damage control surgery) more frequently.

Regardless of age, a therapeutic TAC is ideally performed in cases of abdominal sepsis and/or IAH/ACS even when the underlying disease entities and indications clearly differ according to the child's age (neonatal and infant patients [NEC, meconium ileus/perforation, volvulus, invagination] VS. school-age children and adolescents [perforation, peritonitis, pancreatic]). More often this age-related divergence leads to ACS predisposed entities and allows for a differentiation between neonatal and pediatric risk factors and ACS forms. Tumorous space requirements and bleeding occur in all age groups.

The differentiation between a "prophylactic" and "therapeutic" laparotomy directly affects how intensive medicine is provided and correlates with Bjork et al. 2009 OA classification [56], which applies also to children. In "prophylactic" TACs, the abdominal cavity is usually at least initially not contaminated ("Class A" according to Bjork). However, the peritoneum is usually colonized and/or infected ("Class B") in "therapeutic" TACs. Anti-infection therapy must take the accompanying risk into consideration and be properly adjusted to the bacteria and resistance spectrum as quickly as possible. During this process the prophylactic or therapeutic broad-spectrum antibiosis should be supplemented with a systemic antimycotic (triazole or echinocandin) if the abdomen is expected to be open longer than 3 days. Beyond coding the degree of contamination, the Bjork classification describes the extent of peritoneal adhesion (1°, no adhesion; 2°, incipient adhesion; 3°, enterocutaneous fistula formation; 4°, frozen abdomen). Unlike in the treatment of adults, the latter two only occur as exceptions in pediatric and youth medicine [57].

14.3 Pathophysiology: Pathomechanisms

ACS represents the final lap of an IAH [7]. If diagnosed too late and/or treated inadequately, it can lead to multiorgan failure as well as death. Metabolized products, inflammation mediators, and radicals are released due to local compression, lymphatic and venous stasis, and arterial perfusion deficits with ischemia and possibly reperfusion. At a clinically undetectable point of no return, these elements contribute to self-maintaining hyper-inflammation and explain ACS's high mortality rate (up to 60% and more) [4, 19, 21, 23, 58–60]. Various workgroups found significant changes in concentrations of pro- and anti-inflammatory mediators in connection with IAH and ACS. However, against the backdrop of failing specificity, a therapeutic consequence in the sense of a clinically useful biomarker was not able to be determined.

Thoracic organs, limb muscles, and the brain can be affected by IAH/ACS due to the transdiaphragmal transfer of pressure [61–66]. This is in addition to the damage done to the entire abdominal tissue. Special pathogenic meaning is given to (1) the synthesis of inflammation mediators, which are additionally facilitated by ischemia and reperfusion, and (2) the para- as well as endocrinal, resp. hematogenous and lymphatic, exchange of these mediators between the organs of the large torso, which are highly active as a result of inflammation. It is via the axis of these organs that a self-perpetuating activation of and damage to tissue (lung, liver, and gastrointestinal

tract) can take place before the other organs and tissue are affected by the resulting cytokine storm [30–32]. Pressure, stasis, ischemia–reperfusion, and activation via mediators can accelerate inflammation and the damage to mucosal barrier function of the respiratory and gastrointestinal so much enough that a hematogenic and/or lymphogenic translocation [67–69] of bacteria and fungi occurs [70–78]. This, in turn, leads to sepsis, which can further boost the circulus vitiosus of the systemic hyper-inflammation. Therefore, the gastrointestinal tract is unfairly seen as the "motor of organ failure" [79–81] and should rather be considered part of the "axis of organ failure."

Although opening the abdomen and leaving it open are adequate therapy options to break through circulus vitiosus related to IAH, the process put into motion by the inflammatory cascade following decompression can persist or even be aggravated. This is due to the duration of the pressure damage, resp. the extent and (ir)reversibility of temporarily induced tissue damage and systemic hyper-inflammation. Contributing factors can be hypoxemic metabolic products, mediators (above all tumor necrosis factors, chemo- and interleukins, miRNA), and resident cell surface antigens (selectins, integrins, etc.) [82] that are released from previously poorly perfused organ and tissue sections into the systemic circulation, resp. enable leukocyte extravasation and further boost SIRS, in the context of a reperfusion.

Therefore, after abdominal decompression, intensive care physicians' main tasks are to do the following as quickly as possible:

1. Restore and maintain homeostasis in all organ systems.
2. Recognize persisting or even increased SIRS with capillary leak and fluid diapedesis resulting from ischemia–reperfusion and an additional inflammatory "hit."
3. Recognize a renewed/additional critical increase in IAP (resulting from this "hit" and/or SIRS) with subsequent organ dysfunction (=ACS) in spite of a previously existing OA.

14.4 Organ-Specific Risk Constellations and Their Protection

There are various methods for establishing a temporary abdominal wall closure (e.g., only-skin closure, Bogota pouch, Wittmann Patch, separation, zipper, vacuum pack, VAC® therapy) with several variations and modifications. The incredibly large number is due to the wide range of materials available (e.g., Silastic, absorbable and nonabsorbable artificial mesh, auto-/iso-/homo- and heterologous biomaterials) [36–46, 83, 84]. Aside from nursing aspects, there are no major differences for the intensive care physician in terms of management. In most cases the protective film is changed aseptically, and/or revision assessments are made two to seven times a week. This always includes at least the attempt to reduce the size of the laparostoma, where special attention is paid to the postoperative development of IAP and its consequences following every approximation of the wound margins (see below). In negative wound pressure therapy (NWPT; nowadays the rule), the suction level must be calibrated to suit the patient's age, indication, and possible

complications. Above all, the suction level should usually not exceed −15 cmH_2O (otherwise, −15 bis −50 cmH_2O) in cases of borderline portal vein perfusion (e.g., after a liver Tx) [85]. Possible leakages (e.g., in the cover film) must be stopped immediately to prevent infection or secretion. To ensure there is no secretion resulting from the development of sub-compartments within the abdominal cavity (in spite of vacuum therapy), an ultrasound of all four quadrants should be performed once a day.

14.4.1 IAP Monitoring

It is known that within only a few hours irreversible organ damage and irrevocable inflammatory cascades can start in connection with the dynamic of an IAP increase, resp. an absolute IAH grade [86, 87]. In spite of this and alarming epidemiological data, monitoring IAP is still not a standard part of monitoring in pediatric intensive medicine. According to a survey of pediatric intensive care physicians in Germany, Austria, and Switzerland, only 20% of respondents quantify IAP where needed [17] (additional data not yet published). If there is no method available for continually measuring IAP, the frequency of intermittent IAP measurements should follow a standardized algorithm. Moreover, each pediatric patient's individual risk profile should consider the dynamic of the IAPs previously measured (Figs. 14.1 and 14.2). In the framework of an OA treatment, the basic repertoire of available therapy options does not differ from the general options in cases of IAH and ACS (Fig. 14.3) [7, 17]. The emergency use of nitroglycerin has proven itself in cases of a sudden, massive increase in IAP with potential hemodynamic consequences. Via venal pooling a short-term distribution of volume can be generated and the IAP at least temporarily reduced (e.g., until a decompression operation).

The criteria for a recurrent ACS [7, 17] are fulfilled with a renewed increase in IAP accompanied by organ dysfunction after having performed a therapeutic TAC. In contrast, one speaks of a primary ACS following an increase in IAP with organ damage after having performed a prophylactic TAC. In the latter cases, underlying diseases resulting from a prophylactic TAC and their specific complications as well as the interim interventions and operations performed due to them are to be considered first or second hits whose inflammatory boost can lead to SIRS with IAH and ACS.

In both cases immediate surgical action is necessary. The existing laparostoma is to be expanded so as to normalize IAP as well as blood and lymph flow (at best). Creating or having a laparostoma must not lead to the fatal assumption that the abdomen and organs affected by an IAH are sufficiently relieved by OA management. Especially under the exceptional circumstances of an open abdomen, IAP and pressure capacity estimated using it should be monitored as closely as possible in addition to taking conventional vitals (Table 14.1, Fig. 14.3). Even when the reoccurrence rate for ACS is only 3%, the fatal prognosis, carrying with it an almost 100% mortality rate, should be enough to enable optimal monitoring of the child affected [58].

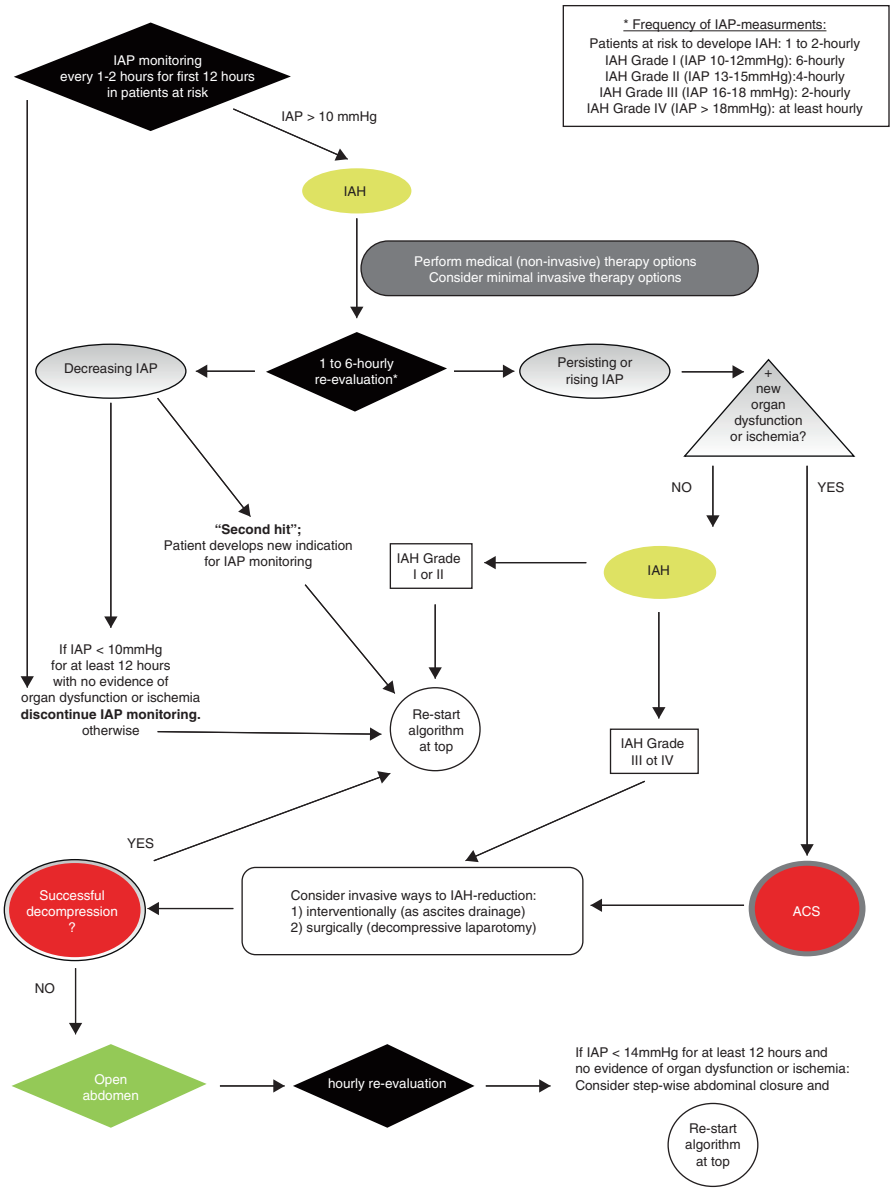
* Frequency of IAP-measurments:
Patients at risk to develope IAH: 1 to 2-hourly
IAH Grade I (IAP 10-12mmHg): 6-hourly
IAH Grade II (IAP 13-15mmHg):4-hourly
IAH Grade III (IAP 16-18 mmHg): 2-hourly
IAH Grade IV (IAP > 18mmHg): at least hourly
IAP monitoring every 1-2 hours for first 12 hours in patients at risk
IAP > 10 mmHg
IAH
Perform medical (non-invasive) therapy options
Consider minimal invasive therapy options
Decreasing IAP
1 to 6-hourly re-evaluation*
Persisting or rising IAP
+ new organ dysfunction or ischemia?
NO
YES
"Second hit";
Patient develops new indication for IAP monitoring
IAH Grade I or II
IAH
If IAP < 10mmHg for at least 12 hours with no evidence of organ dysfunction or ischemia discontinue IAP monitoring. otherwise
Re-start algorithm at top
IAH Grade III ot IV
YES
Successful decompression ?
Consider invasive ways to IAH-reduction:
1) interventionally (as ascites drainage)
2) surgically (decompressive laparotomy)
ACS
NO
Open abdomen
hourly re-evaluation
If IAP < 14mmHg for at least 12 hours and no evidence of organ dysfunction or ischemia:
Consider step-wise abdominal closure and
Re-start algorithm at top

Fig. 14.1 Pediatric diagnostic and therapy algorithm for monitoring and treating IAH/ACS in accordance with the child's individual risk profile and most recently measured IAP

Fig. 14.2 Depiction of an intraoperative situs during a revision operation due to recurrent ACS in spite of open abdomen management on the floor of a Grade IV fulminant progressive neuroblastoma (prior to chemotherapy). As a result of a renewed increase in IAP, the abdominal organs are being compressed and pushed into a wave-shaped abdominal wall mesh (see picture). Also noticeable is the surface coloring of the less perfused liver and edematously distended intestinal loops. The child died shortly after the operation due to cardiovascular failure that could not be remedied

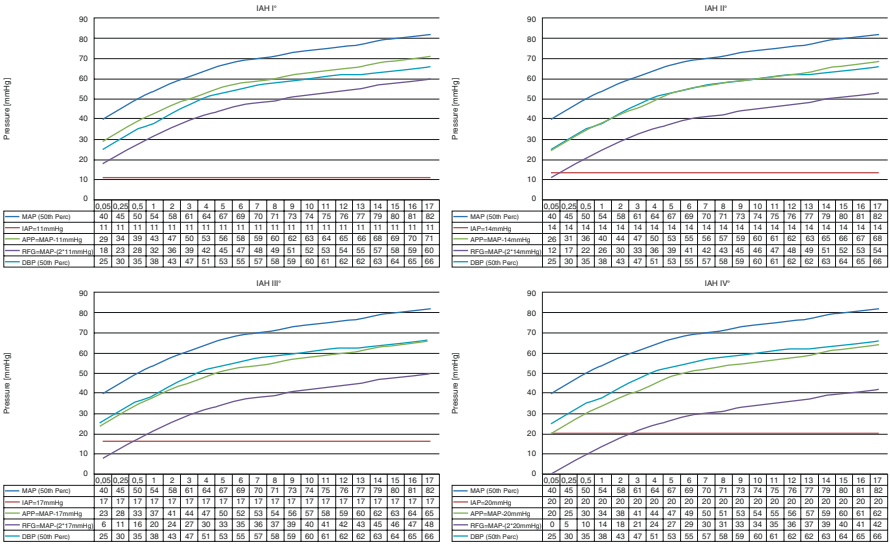

Fig. 14.3 Influence of IAP on abdominal perfusion pressure (APP) and renal filtration gradient (RFG) in children and adolescents. The 50th percentile of standard values for mean arterial pressure (MAP) and diastolic blood pressure (DBP) according to age (in years) as well as the influence of different IAH grades on the resulting APP, resp. RFG, which can already fall below the age appropriate diastolic level during a Grade I IAH. As of a Grade II IAH, the APP level for all age groups lies either at or below the diastolic level, which can result in parenchymatic stasis due to a missing drop in pressure. In regard to the kidneys, weakening diuresis can be explained by this

14.4.2 Cardiocirculation

Depending on the grade of IAP, blood reserves from venous pooling areas in the abdomen can become mobilized, and cardiac output appears normal for a short period. In adults one observes this effect, known as autotransfusion, at IAP levels of around 15 mmHg [88, 89]. The percentiles for this threshold have not been established for children but would correspond with IAH Grade I. If the pooling reserves are used up, resp. if there is an IAP increase surpassing this threshold, there can be a sudden drop in cardiac output with arterial hypotension [90–93]. The main cause of this could be pressure-induced venous congestion [92, 94]. Another possibility is a direct compression of first lymphatic, venal, capillary, and later arterial vessels [94–99]. A result of this is reduced returned venal flow in the right ventricle. This is not necessarily reflected in decreasing central vein pressure (CVP), which is why CVP generally fails as a volumetric parameter in cases of IAH/ACS [90] [100–102]. Interpreting the so-called filling pressure is further impeded by an OA therapy with an NWPT set according to indication and patient age. Under IAH, ACS, and OA circumstances, extended hemodynamic monitoring would be desirable [103]. Pulmonary arterial catheters and dilution technology are only justifiable in older children, though, in the context of size-related limitations. Processes like impedance cardiography (electrical velocimetry) [104] and somatic near-infrared spectroscopy [105–107] will increasingly play a role as alternatives to noninvasive assessments of macro- and microcirculation.

The bi-ventricle functional limitation observed in IAH/ACS is explained by direct myocardium compression (occurring when the diaphragm is elevated) [108], the cardio-depressing effect of mediators circulating (above all TNFα) [109, 110], and increased peripheral resistance (afterload) [111]. Aside from measuring blood pressure invasively, echocardiography is the method of choice for assessing hemodynamic, including volume status and contractility [112]. Figure 14.3 depicts the influence of increasing IAP on the residual perfusion of abdominal tissue and the transition to ACS that is often observed starting at IAH Grade II.

In addition to multimodal options for reducing IAP (Table 14.3), stabilizing blood pressure ensures sufficient abdominal perfusion in accordance with APP = MAP−IAP (analogue to cerebral perfusion pressure with CPP = MAP−ICP; synonymous, splanchnic perfusion pressure) [7, 113–115]. This can be achieved above all by optimizing the volume status and use of catecholamine [116, 117].

Besides visually assessing ventricular filling to estimate the volume required, one must balance all output on an hourly basis. This includes losses via the abdominal aperture and, where necessary, other drainages (e.g., pleura drainage) as well as perspiration. Secondary (conventional clinical) criteria for identifying a relative hypovolemia are:

- Responsiveness to liver palpation
- A swing in the arterial pressure curve [118]
- Blood gas analytical signs of a metabolic acidosis (pH, base excess, lactate),
- Inferior vena cava diameter before its junction with the liver veins (sonography) [112]
- A central venous saturation (CVS) <70% (insofar there is no shunt vitium or parenchymatous lung disease)

Against the backdrop of an expected post-decompression (resp. post-OA operation) cytokine storm, within the first 24–48 (−72) h, a harmonious or positive balance is the rule in order to maintain sufficient macro- and microcirculation. Lost fluids must initially be replaced hourly to achieve a "continuous balance." In addition to full electrolyte solutions, colloidal volume replacement like freshly frozen plasma (INR > 1.5–2.0 or bleeding tendency) and human serum albumin should be used as needed (targets, serum total protein >40 g/L, resp. serum albumin >20 g/L). The balancing of the required quantity and decision for or against a crystalloid or colloidal volume replacement must, on the one hand, involve the loss of fluid over the open abdomen and, on the other hand, the possibly already existing overflow of the lung. Existing restrictions on the function of certain organs or systems should also be taken into consideration, thus combining fluid substitution and the replacement of missing substrates. For example, a substitution of albumin or GFP should be considered for hepatic dysfunction, and erythrocyte and thrombocyte transfusions may be useful in bone marrow depression [119, 120]. However, it must also be noted, that for rheological reasons transfusion of concentrates of erythrocytes and thrombocytes must remain under special limits (target Hb, 8–10 g/dL; target thrombocytes, >50,000/μL [without bleeding]) following Txs. When there is cyanotic vitium, an Hb level of 12 g/dL should not be surpassed; otherwise, the usual transfusion limits apply. Normally starting on the third day following a laparostomy (at the latest), a negative balance should be possible with the lessening SIRS, where the goal is reaching the patient's preoperative body weight. Without a negative balance, it is impossible to close the abdominal wall without reraising IAP [7, 121].

Considering the hemodynamics, systemic inflammation, and inevitable deep analgosedation, catecholamine therapy is usually unavoidable. To the benefit of certain substances, the selection of appropriate catecholamines in pediatrics still happens without sufficient evidence. In neonatology, dopamine, dobutamine, and dopexamine are used most; in pediatric intensive care stations, norepinephrine and—where necessary—epinephrine are commonly used. The extent of cardiac functional limitations detected by echocardiography and/or that of septic disease components determines the choice of dobutamine and/or norepinephrine; epinephrine is used in cases of uncontrollable circulatory insufficiency. When this is accompanied by adrenal insufficiency, hydrocortisone should act as a temporary substitute. Thus far there has been no evidence of the use of phosphodiesterase inhibitors. Whether they can contribute to a better outcome due to their inotropes and vessel dilating properties needs to be determined soon within the scope of studies [122].

14.4.3 Kidney Function and Fluid Balancing

The three clinical cardinal symptoms of an ACS are (1) cardiocirculatory, (2) respiratory impairment and/or failure [123], and (3) oliguria, resp. anuria [124–128]. Figure 14.3 depicts IAP's dependency on APP and RFG. As hemodynamics stabilize, diuresis performance can improve—even with high IAP levels. At least once a day, the duplex sonographic flow pattern of the kidney arteries and parenchymatic

perfusion should be assessed along with ruling out thrombosis of the kidney veins (resulting from pressure and stasis) [129]. When there is end-diastolic zero, negative, or oscillatory flow, the efficiency and extent of the OA should be viewed critically together with the IAP dynamic [130].

In addition to optimizing IAP and hemodynamic (see above), consistent use of diuretics (as needed in combination with human serum albumin) enables a negative balance to be achieved following the initial acute phase. Should the use of hydrochlorothiazide, spironolactone, loop diuretics, and etacrynic acid not suffice, then theophylline, volume restrictions, and dialysis should be considered.

The exception: For rheological reasons, diuretics can only be prescribed cautiously following liver Txs.

14.4.4 Respiration

Autonomous respiration without the help of a respirator cannot be realized with an open abdomen—even when there is a loss of stability in pediatric patients. Risks for developing acute respiratory distress syndrome (ARDS) [131, 132] are:

- (Long-term) respiration
- Atelectasis formation due to an IAH-induced elevated diaphragm (above all dorso-basal)
- Limited ability to mobilize due to laparostomy
- Inflammatory stimulation of a lung that has already been activated by circulating mediators

To counteract an elevated diaphragm and the development of pressure-induced dystelectasis, the best PEEP principle or a balancing of PEEP with the current IAP (in the sense of compensatory counterpressure) has been suggested repeatedly for adult patients in various workgroups [133–138]. Although there is absolutely no evidence supporting this for pediatrics, relatively high PEEP values between 8 and 15 cmH_2O (depending on age and clinic) are the norm in pediatric cases of IAH/ACS/OA. Aside from regular blood gas analyses and X-rays, daily ultrasound examinations of the thorax make sense in order to estimate PEEP requirements, because poorly aerated lung areas can be recognized more sensitively and specifically via sonography [139]. Moreover, pleural effusion expected in connection with SIRS can be assessed and treated generously with a puncture or drainage. Through this vital capacity limited by an IAH can be increased and, in doing so, potentially deescalate the invasiveness of mechanical ventilation. IAP can also be measurably reduced by transferring pressure transdiaphragmally [140].

If blood gas analysis, clinical, and radiological results point to a transition toward ARDS, the ARDS Network's [132] recommendations regarding oxygen and PEEP support are to be followed (Table 14.4, including ARDS definition). Attempting to increase PEEP during IAH should be done carefully and in consideration of the fact that every further increase in intrathoracic pressure can lead to a potentially acute limitation of the initial load and hemodynamic destabilization of the cardiac pump

Table 14.4 ARDS net protocol for ventilation optimization in shock lung/ARDS [156]

FiO_2	0.3	0.4	0.5	0.6	0.7	0.8	0.9	1.0
PEEP [cmH_2O]	5	5–8	8–10	10	10–14	14	14–18	18–24

According to the Horowitz index (PaO_2/FiO_2), there are three ARDS grades (I.°, <300; II.°, <200; III.° < 300). Additional diagnosis criteria are bilateral infiltration and ruling out pulmonary hypertension via echocardiography. In accordance with the respective oxygen needs, an increase in PEEP applying the values in the table should be tested

function. This also applies for recruitment maneuvers for reopening dys- or atelectatic lung areas [141]. For high respiration levels, resp. PEEP levels, to work, it is generally necessary to have a sufficiently large nasopharyngeal tube (usually 0.5 mm more in circumference than the norm for the respective percentile) and a (micro-)cuff. In this situation it is also necessary to regularly ensure air is not trapped and the intrinsic PEEP is not increasing.

Since the probability of lung alteration combined with all potential risk factors is very high, every further noxious agent, for example, a shearing trauma, must be avoided when attempting to create low tidal volume ventilation (5–7 mL/kgKG/min) for lung protection. The overwhelming majority of patients affected can be sufficiently oxygenated and ventilated conventionally (as needed also accepting a potential permissive hypercapnia (target, pH > 7.2 and plateau pressure < 30 cmH_2O)) [142]. Only in special cases must nitrogen monoxide, high-frequency oscillation (HFO), or even extracorporeal oxygenation be considered (and critically questioned) [143, 144].

Due to the open abdomen, patient mobility (including the prone position) is limited [145]. Because of this, consistent inspiratory gas conditioning and warming, regular inhalation therapy with mucolytic and—where necessary—medicine dilating the bronchial tubes, and stringent physiotherapeutic secretion mobilization take on prophylactic and therapeutic importance. With potential bacteria translocation in mind, regular microbiological examinations of tracheal secretion should occur and antibiosis adjusted early on.

14.4.5 Supportive Therapy

In IAH Grade I, deep analgosedation [146] (according to WHO recommendations) can suffice; however, in IAH Grade II (at the latest) and when there is a laparostoma, additional relaxation must be considered. In terms of more pronounced metabolization, there is an earlier adjustment in children and adolescents than in adults. This makes it necessary to regularly change the analgosedativa schemata with additions and changes in substance groups (including benzodiazepine, opioids, barbiturates, α_1-agonists, dexmedetomidine, low-potency neuroleptics, antidepressants). Depending on the necessary length of the laparostoma therapy, this can even turn into off-label use of inhalative anesthetics (e.g., iso-/sevoflurane) via a vaporization system (AnaConDa®) [147, 148] that can be combined with conventional respirators.

Although medicinal (e.g., macrogol) and interventional purgative measures (e.g., gastric tube, intestinal tube, endoscopy) for reducing intraluminal volumes are in the foreground of conservative IAH therapy (Table 14.3), minor

mucosal feeding of intestinal villi should at least be attempted [149–153]. Laying a jejunal and duodenal tube as well as regularly administering enemas and prokinetics (Naloxon, Metoclopramide, Erythromycine [154]) have proven themselves as methods for overcoming gastroparesis and enteric paralysis, which frequently accompany and are exacerbated by the combination of medicines mentioned above. However, erythromycins [154] interact with various immunosuppressants and are, therefore, contraindicated following a transplantation.

Above all, the vast majority of energy required must initially be covered parenterally. In doing this, the composition of the total or partial parenteral nutrition must be adjusted to the energy needs of the individual patient age and post-aggression stage. Protein and fat can be metabolized 2–4 days after an operation (at the earliest). Micronutrients and vitamins should be substituted starting on the fifth to seventh post-OP day. Deficiencies and insufficient substitution are monitored weekly via enhanced laboratory monitoring [155]. Various workgroups have proclaimed the positive effect of enriching glutamine, selenium, zinc, as well as immuno-nutrition on the ICU outcome in adults; yet there is absolutely no evidence of this in children.

Take-Home Messages

- In children and adolescents, performing prophylactic laparostomata predominates in connection with organ transplantation and abdominal wall closures.
- An IAH and ACS can (re)occur anytime following a TAC, resp. incremental laparostoma reduction. Therefore, conventional intensive care monitoring of cardiac circulation, respiration, and balance is to be supplemented with close IAP monitoring, regular sonographic and echocardiographic examinations, as well as expanded hemodynamic monitoring (if possible).
- Therapeutic measures enabling prompt closure of the abdominal wall equal those for prophylaxis and treatment of IAH.
- Adequately caring for children and adolescents with an open abdomen requires the provision of differing concepts for apparative organ replacement in case of secondary (multi)organ failure and/or septic complications.

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Nutritional Support in Patients with an Open Abdomen

15

Patricia Marie Byers and Andrew B. Peitzman

Key Points (Fig. 15.3)

- Start enteral feedings in all open abdomen patients with intestinal continuity as soon as they are hemodynamically resuscitated.
- Plan for post-pyloric enteral access at damage control follow-up laparotomy.
- Start TPN if at least 60% of nutritional requirements are not met by day 4.
- Use an enteral therapy bundle and volume-based ordering to maximize success with enteral feeding.
- Pay careful attention to protein support and micronutrient replacements.
- Try to enterally feed fistula patients as soon as possible, but start TPN after resuscitation is complete with hypocaloric high-protein therapy.

15.1 Metabolic Stress in the Critically Ill Patient with an Open Abdomen

Our understanding of the metabolic response to critical illness and the response to the open abdomen has evolved over the last several decades. These patients with an open abdomen are highly catabolic. Malnutrition in this setting is defined by

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nutrient deprivation coupled with a metabolic disturbance that causes increased protein turnover with a rapid loss of lean body mass. Host defenses are compromised with poor wound healing, increased infection rates, prolonged ileus, lengthened hospital stay, and increased mortality. It is important to understand the metabolic phases of injury of this catabolic response to customize the optimal nutritional support for each phase.

The phases of damage control laparotomy coincide with the phases of the metabolic response as outlined by Cuthbertson in the early 1930s [10–12]. The initial operative phase of damage control laparotomy and the following ICU resuscitation coincides with the ebb phase of injury. This immediate response to tissue injury is fueled by catecholamines with hemodynamic and reperfusion disturbances characterized by a pronounced acute phase reaction with vasoconstriction. Optimally, within 12–24 h, this phase is completed with normalization of perfusion, core temperature, and resolution of lactic acidosis.

The flow phase follows, and the metabolic environment changes, now with increased levels of catecholamines and cortisol, usually persisting from 3 to 21 days [11]. There is a state of increased energy expenditure and hypercatabolism with protein turnover and muscle protein breakdown for substrate, along with increased cardiac output and oxygen consumption. This "auto-cannibalism" can be viewed as an adaptive response that provides the brain and injured tissues with substrate to promote healing. Insulin resistance is responsible for the decreased peripheral use of glucose and the increased rates of lipolysis and proteolysis for the provision of amino acids and fatty acids as fuel substrates. The conversion of peripherally mobilized amino acids (primarily alanine), lactate, and pyruvate to glucose by gluconeogenesis is not suppressed by hyperglycemia or the infusion of glucose solutions in this catabolic state. Branched-chain amino acids are used preferentially as fuel in the skeletal muscle. There are some amino acids that are taken up selectively by tissues for specific purposes. For example, glutamine, a conditionally essential amino acid, is taken up by the proximal nephron to sustain ammoniagenesis and to counteract acidosis, by fibroblasts and enterocytes to promote healing and by immune cells for replication [13]. While adipose tissue is expendable and can be utilized as fat calories, protein is not, as all proteins have either structure or function [6].

If the stress state is prolonged, the amino acid pool will become depleted of essential amino acids, and protein synthesis cannot match the increased rate of muscle protein breakdown. Over time, there will be functional deterioration in organ system function with poor wound healing, atelectasis, pneumonia, prolonged ventilator dependence, and compromised host barrier function. This will prolong the stress state and result in poor outcomes, long-term functional disability, and increased mortality [14, 15]. This condition may be exacerbated by the development of anabolic resistance [12] and is seen in the newly recognized syndrome of persistent inflammatory catabolic syndrome (PICS) that appears to be more pronounced in older patients [16] (Table 15.1).

Table 15.1 Persistent inflammatory catabolic syndrome

Diagnosis	Treatment	Outcome
Ongoing protein catabolism	High-protein nutritional support	Unsuccessful therapy results in late deaths in chronic facilities, often following ICU discharge
Low serum protein markers		
Muscle wasting/debilitation		
Poor wound healing	Aggressive daily physical therapy	
Elevated WBC counts		
Lymphocyte depletion with TLC < 800/mm^3		
Apparent immunosuppression with recurrent infections	Anabolic agents when indicated	
Inflammatory state with elevated IL-6, CRP > 150 μg/dL		

Adapted from Gentile LF, Cuenca AG, Efron PA, et al. Persistent inflammation and immunosuppression: a common syndrome and new horizon for surgical intensive care. J Trauma Acute Care Surg. 2012;72(6):1491–1501

15.2 Diagnosis of Nutritional Risk in Critically Ill Patients

Assessment of nutritional risk may be overlooked as attention is drawn to the therapy of shock, respiratory failure, or sepsis. However, it is important to identify patients with the highest risk early, as they have the worst outcomes and benefit the most from nutritional interventions [17–19].

Patients with an open abdomen may enter the illness malnourished, adequately nourished, or obese. The preexisting state of health of the patient and the presence of comorbidity also contribute significantly. The open abdomen itself is a major contributor to nutritional risk. Nutritional risk scores have been developed that take into account baseline nutritional status, health status, inflammation, and severity of illness. The two scores that have been recommended by the most recent guidelines include the Nutritional Risk Score (NRS) 2002 and the NUTRIC Score [18, 19].

The NRS [18] is easiest to calculate and uses disease severity and a history of previous malnutrition or recent poor intake to calculate risk (Table 15.2). It requires previous weight loss history and recent dietary to calculate, so that it may be difficult to use in trauma and emergency surgery patients. A score >3 signifies risk, and a score >5 signifies high risk.

The NUTRIC Score has been validated in critically ill patients [19] (Table 15.3). It requires the calculation of APACHE and SOFA scores and, thus, is primarily based on disease severity. A score of >5 defines a high-risk patient. An assessment of nutritional risk should be performed on every patient with an open abdomen upon entering the intensive care unit to determine the approach for nutritional support.

A patient may enter into a course of illness with an open abdomen with micronutrient deficiencies. In addition, micronutrient deficiencies can occur rapidly in these patients due to increased utilization, compartment shifts, and losses in peritoneal fluid [20]. Vitamin and mineral deficiencies may be associated with

Table 15.2 The nutritional risk score (NRS 2002)

Nutritional risk screening 2002 (Espen Guideline)			
Impaired nutritional status		Severity of disease	
Mild—score 1	Weight loss >5% in 3 months or food intake <50–75% in preceding week	Mild—score 1	Hip fracture, chronic patients with acute complications: cirrhosis, COPD, ESRD, DM, malignancy (oncology)
Moderate–score 2	Weight loss >5% in 2 months or BMI 18.5–20.5 and impaired general condition or food intake 25–50% in preceding week	Moderate—score 2	Major abdominal surgery, stroke, severe pneumonia, malignancy (hematology)
Severe—score 3	Weight loss >5% in 1 month or >15% in 3 months or BMI < 18.5 and impaired general condition or food intake 0–25% in preceding week	Severe—score 3	Head injury, bone marrow transplant, ICU patients (APACHE II > 10)
Score:		Score: Age adjustment score: 0–1 Total score: _____	
Find the highest score for impaired nutritional status and add it to the highest score for severity of disease; then if age ≥ 70 years, *add 1* to get the total score			

COPD chronic obstructive pulmonary disease, *ESRD* end-stage renal disease, *DM* diabetes mellitus, *APACHE* acute physiology and chronic health evaluation, *BMI* body mass index in kg/m^2
Adapted from Kondrup J, Rasmussen HH, Hamberg O, Stanga Z, and an Ad Hoc ESPEN Working Group. Nutritional risk screening (NRS 2002): a new method based on an analysis of controlled clinical trials. Clin Nutr. 2003;22(3):321–336

Table 15.3 The revised NUTRIC Score

Variables in NUTRIC Score	Range	Points
Age	<50	0
	50–74	1
	≥ 75	2
APACHE II	< 15	0
	15–<20	1
	20–28	2
	≥ 28	3
SOFA	<6	0
	6–<10	1
	≥ 10	2
# Comorbidities	0–1	0
	2+	1
Days from hospital to ICU admit	0–<1	0
	1+	1
		Total=

APACHE II acute physiology and chronic health evaluation, *SOFA* sequential organ failure assessment
Adapted from: Heyland DK, Dhaliwal R, Jiang X, Day AG. Identifying critically ill patients who benefit the most from nutrition therapy: The development and initial validation of a novel risk assessment tool. Critical Care. 2011;15(6):R268

end-organ dysfunction and may affect the response to sepsis and nutritional support. Testing for serum levels of these micronutrients currently is the only clinically available tool.

15.3 Nutritional Therapy

15.3.1 Dosage, Timing, and Route of Nutrients

Nutritional goals for critically ill open abdomen patients may be calculated utilizing custom formulas such as the Harris–Benedict equation, indirect calorimetry, or fixed weight-based formulas. Although easy to use, it is important to note that fixed weight-based formulas are incorrect in 70% of ICU patients, as they do not account for changes in requirements associated with age and gender. This issue has gained importance with the recent appreciation of the narrow range of optimal nutritional support needed to avoid underfeeding and overfeeding.

Indirect calorimetry yields the most accurate information regarding an individual patient's energy utilization but still requires interpretation regarding therapeutic goals. Ventilator support, renal replacement therapy, and pain issues can interfere with results. In the first week following treatment with an open abdomen, a conservative interpretation would seem to be best. For example, the Harris–Benedict equations can be utilized and set at basal to 1.3 times basal to meet the requirements during this period of time if indirect calorimetry is not available (Table 15.4).

In addition to calculating caloric requirements, the composition of the macronutrients must be considered. Excess carbohydrates should be avoided as they can cause problems with blood sugar and carbon dioxide production, which can be associated with increased complications and ventilator days. Intravenous dextrose should not be increased until blood sugars are under adequate control. If there are concerns regarding carbon dioxide production, carbohydrates should be limited to 4 mg/kg/min. It is also important that all dextrose in intravenous fluids and all fats in lipid-based medications administered be quantified and their calories counted as support received. Intravenous fat calories should be limited to 20–30% of total calories, with more stringent restrictions during the first week.

Recent studies focused on protein administration have suggested that protein support may be paramount early in critical illness and may require a separate analysis [21]. Daily protein requirements in these patients are high, in the range of 2–2.5 g/kg/day. Nitrogen balance studies should be performed to accurately assess the protein needs of these hypercatabolic patients with excessive protein losses. A minimum of 2 g of nitrogen per liter of abdominal fluid drainage should be added to

Table 15.4 Harris–Benedict equations

BEE Women = 655 + (9.6 × weight in kg) + (1.7 × height in cm) − (4.7 × age in years)
BEE Men = 66 + (13.7 × weight in kg) + (5 × height in cm) − (6.8 × age in years)

BEE Basal energy expenditure

Table 15.5 Nitrogen balance equations

Nitrogen (N) balance = N (in) − N (out)
N (in) = protein/6.25 (g/day)
N (out) = TUN[a] (g/day) + gastrointestinal losses (2 g/day) + cutaneous losses (0–2 g/day) + abdominal fluid loss (2 × Liters abdominal fluid loss g/day) + fistula loss (2 × Liters fistula effluent g/day)

Adapted from Polk TM, Schwab CW. Metabolic and nutritional support of the enterocutaneous fistula patient: a three-phase approach. World J Surg. 2012;36:524–533

[a]TUN (Total urinary N) can either be measured directly or estimated by measuring urine urea N and dividing by 0.8

nitrogen losses in these patients, while the abdomen remains open [22]. An additional gram of nitrogen losses should be added for every 500 mg of succus lost from the abdominal fistulae [23] (Table 15.5). Achieving both protein and caloric goals decreases mortality in intensive care unit patients [6].

Calculating nutritional requirements in the morbidly obese patient with an open abdomen is especially challenging. Lean body mass must be maintained to promote wound healing without overfeeding. Guidelines from the American Society of Enteral and Parenteral Nutrition and the Society of Critical Care Medicine advocate hypocaloric, high-protein nutritional support in these patients [17]. Caloric support of only 50–70% of predicted energy needs from standard equations or 14 kcal/kg of actual body weight has been proposed. Protein support is recommended at 2.5 g/kg of ideal body weight [24]. Due to the risk of underfeeding with this strategy, monitoring of nutritional status and response to the support, such as wound healing, is critical.

It is also important to be mindful that caloric requirements may increase as deficits are created. After 7–14 days, as patients progress to a more "chronic" critically ill phase, and develop PICS, they will require increased caloric support at 1.5 times the resting measurement or basal calculation to reduce caloric deficits and diminish adverse outcomes. Furthermore, patients who develop secondary complications, such as fistulae, will need increased protein support. Protein administration should not be restricted in patients who develop acute kidney injury. This complication is catabolic and should be treated with renal replacement therapy as needed.

Feeding during the ebb phase of injury during the critical period of resuscitation is not indicated in most patients. However, within 24–48 h of admission, once hemodynamic stability and resuscitation have been achieved, enteral feedings should be started in patients with an open abdomen with gastrointestinal continuity. In general, do not initiate enteral nutrition in trauma and surgical patients on vasopressor support [25]. However, in select circumstances, trophic feedings may be administered via the gastric route to patients who are weaning from vasopressor therapy [17, 26]. If there is gastric hypoperfusion, feedings will not be tolerated and should be stopped. This recommendation is based on the fact that the stomach is a sensitive monitor of gastrointestinal hypoperfusion and that the majority of cases of intestinal necrosis with enteral feedings have occurred with jejunostomy feedings [25].

Early enteral feedings should always be attempted in patients with an open abdomen because they promote gut-mediated immunity, maintain microbial diversity, increase intestinal blood flow, and attenuate the hyperdynamic stress response associated and immune suppression seen in these patients [2, 4]. Early enteral feedings are associated with increased rates of primary fascial closure, lower fistula rates, and lower hospital charges [3]. Animal studies have demonstrated increased mucosal permeability with tight junction damage when the small intestine is exposed to air [27]. These injuries were reversed when enteral nutrition was provided. Enteral nutrition decreases gut weight and improves intestinal perfusion, lymph flow, and venous return, thus decreasing edema and increasing the possibility of abdominal closure [28, 29]. The Western Trauma Study [1] and Dessanaike et al. demonstrated similar benefits in open abdomen patients without bowel injury [4]. However, the same benefits failed to occur in patients with bowel injury [1]. Byrnes et al. demonstrated an association of delayed enteral feeding in patients with bowel injuries [5]. This delay in adequate enteral feeding of this subset of patients may explain the lack of benefits demonstrated in the Western study [1].

To maximize the opportunity for successful enteral feeding, a nutritional bundle should be implemented [17]. This ensures that there is the same diligence in accomplishing enteral feedings as is routinely utilized in other treatment modalities employed in critical care [17] (Table 15.6). Daily assessments need to be performed along with an aggressive approach for enteral access. Plans for enteral access should be made intraoperatively during intra-abdominal procedures performed subsequent to the index damage control case. Post-pyloric access will be needed whenever gastric ileus inhibits reaching the goals of therapy. Bedside nurses must be empowered to participate in feeding decisions and metabolic monitoring [26]. Additionally, the entire physician team will need to endorse this clinical approach.

Management becomes more challenging when enteral feeding is impossible due to intestinal discontinuity or unsuccessful due to intestinal injury. If calculated enteral goals cannot be met, protein modules can be added to meet protein requirements. A recent study advocating permissive underfeeding during the first 2 weeks of intensive care unit presentation reported no difference in survival between

Table 15.6 Enteral feeding bundle

Assess nutritional risk immediately upon ICU admission
Calculate energy and protein requirements separately—monitor daily
Initiate enteral feeding within 24–48 h of presentation
Increase enteral feeding to goal as soon as possible
Take measures to reduce the risk of aspiration
Attempt post-pyloric tube placement and improve tolerance as needed with prokinetic agents
Implement enteral feeding nursing protocols and use volume-based nutritional prescriptions replacing missed feedings due to procedures
Do not use gastric residual volumes as part of routine care

Adapted from McClave SA, Taylor BE, Martindale RG, et al. Guidelines for the provision and assessment of nutritional support therapy in the adult critically ill patient: Society of Critical Care Medicine (SCCM) and American Society of Parenteral and Enteral Nutrition (ASPEN). J Parenter Enter Nutr. 2016;40(2):159–211. Permission requested

administration of 50 versus 70% of the calculated caloric requirements as long as equal protein support between 55 and 60 g/day was achieved [30]. The limitation of this study is that both groups were relatively underfed for both calories and protein, making interpretation of the results difficult.

Calories added from intravenous fluids and lipid-based medications should be calculated as supplemental calories and should meet at least 60–80% of calculated goals. When at least 60% of calculated goals cannot be met utilizing the above strategies, it must be decided when to add supplemental parenteral support. There has been considerable controversy surrounding the utilization of supplemental parenteral support in critically ill patients. Multiple trials published since 2011 have demonstrated conflicting results.

The TICACOS trial reported increased ventilator days and complications in patients receiving supplemental parenteral nutrition. However, in this trial, the patients were overfed as they failed to account for energy delivered from other therapies such as lipid-based sedation and dextrose solutions utilized for drug delivery [7]. The EPaNIC trial, the largest ICU nutrition trial to date, again demonstrated that early aggressive nutritional support with supplemental parenteral nutrition resulted in more infectious complications and prolonged ventilator days. This trial utilized generous equation-based energy targets and initiated early supplemental parenteral nutrition on day 1 versus waiting until day 7 [8]. Both studies essentially confirmed that early overfeeding is detrimental when started too early, especially in patients with minimal nutritional risk. This is in contrast to the trial by Doig [31], demonstrating that supplemental parenteral nutrition was beneficial when started on day 3–4 with lower caloric goals. Other studies have demonstrated increased complications and mortality risk when parenteral nutrition is withheld in patients with high nutritional risk [32–34]. Another study of critically ill patients with prolonged ventilator support demonstrated that patients with low nutritional adequacy during the first weeks of illness had a decreased survival time at follow-up and lower functional status at 3 months [35]. This suggests that inadequate nutritional support in the early phases of critical illness has an adverse impact on patient outcomes.

The timing and indications for combination therapy must be determined based on the individual patient's baseline nutritional status, the presence of intestinal injuries and continuity, the presence of intestinal fistulae, and the anticipation of operative interventions and potential abdominal closure. As parenteral nutrition may be lifesaving in high-risk patients, early support on day 3–4 should be considered when enteral feedings are not feasible or successful [31]. Calories and protein from enteral support that is tolerated and calories from dextrose and lipid emulsions that are administered should be included when calculating additional parenteral support needed to reach 80% of goals. It would be unusual for a standard parenteral formula to be used in this setting without resultant caloric overfeeding. Customized hypocaloric high-protein formulas are most appropriate, attempting to achieve at least 2–2.5 g/kg of protein daily.

Nonprotein calories can be given as dextrose or via lipid emulsions. In 1962 Wretland developed long-chain triglyceride emulsions from soybean oil, using egg yolk phospholipid as the emulsifier. Recently, new lipid formulations with fatty acid

profiles that are less inflammatory have been developed, are now available, and may benefit this patient population [36].

Micronutrient deficiencies described in the 1970s led to the development of balanced vitamin and trace minerals for parenteral nutrition solutions. However, these solutions were developed to prevent deficiencies in stable home parenteral nutrition patients and have not been specifically adapted to meet the needs of critically ill patients [37] (Table 15.7). Losses of vitamins and minerals associated with the open abdomen increase these requirements further [20]. Hourigan et al. demonstrated significant losses of vitamins A, C, and E, as well as zinc, iron, and copper from peritoneal fluid in a series of open abdomen patients (Table 15.7). Care must be taken in the repletion of these vitamins and minerals due to the potential prooxidant characteristics of copper, selenium, and iron that may occur with doses that exceed repletion. For example, selenium has been shown to have a potentially therapeutic role in the systemic inflammatory response syndrome (SIRS) and other critically ill patients, but it was detrimental when given in large doses in the MetaPlus study [38]. Zinc, selenium, and vitamins should be administered in physiologic doses until further studies are done to determine whether there is indication or safety in administering pharmacologic doses.

Table 15.7 Vitamin and mineral requirements (EC = enterocutaneous; EA = enteroatmospheric)

Vitamin or mineral	Daily requirement	Extra losses of open abdomen	Extra losses of EC/EA fistula
Biotin	60 mcg		
Chromium	10–20 mcg		
Copper	0.1–0.5 mcg	*	*
Folic acid	600 mcg		*
Iron	0–2 mg	*	
Niacin	50 mg		
Pantothenate	15 mg		
Pyridoxine	5 mg		
Riboflavin	5 mg		
Selenium	20–200 mcg		
Thiamine (B1)	5 mg		
Vitamin A	2500 IU	*	
Vitamin B12	12 mcg		*
Vitamin C	1000 mg	*	*
Vitamin D	25–100 mcg		
Vitamin E	50 IU	*	
Vitamin K	1–2 mg		
Zinc	1–15 mcg	*	*

* extra losses of the indicated element

Adapted from: From Byers PM, Hameed SM, Dudrick SJ. Preoperative and postoperative nutritional support: strategies for enteral and parenteral therapies. In: Asensio JA, Trunkey DD, editors. Current therapy of trauma and surgical critical care. 2nd ed. Philadelphia: Elsevier; 2016. p. 736. Hourigan LA, Omaye ST, Keen CL. Vitamin and trace element loss from negative pressure wound therapy. Adv Skin Wound Care. 2016;29 (1):20–25. Polk TM, Schwab CW. Metabolic and nutritional support of the enterocutaneous fistula patient: a three-phase approach. World J Surg. 2012;36:524–533

The amino acid deficiency of glutamine, and its conditionally essential status in critically ill patients, was addressed with the development of the L-alanine-L-glutamine dipeptide solution in the early 1980s by Furst et al. [39]. However, the instability of this formula, its cost, volume requirements, and controversial indications have prevented its routine use. The supplemental use of parenteral glutamine in critically ill patients has been the object of considerable debate. Glutamine becomes the primary fuel of the intestinal mucosa during stress and, as a consequence, contributes to intestinal villous integrity. Glutamine is not stable in solution and therefore cannot be a component of parental amino acid formulations. For this reason, it must be administered separately as L-alanyl-L-glutamine dipeptide. Smaller studies in single centers performed on surgical and trauma patients have demonstrated positive results in survival and a beneficial effect on glucose homeostasis [40]. However, more recent large multicenter studies performed in mixed patient populations with medical patients have demonstrated adverse outcomes when supra-therapeutic doses of glutamine were administered [38, 41]. Most recently, a multicenter trial performed in surgical patients (gastrointestinal, vascular, and cardiac) without renal or hepatic impairment demonstrated neither benefit nor harm with glutamine supplementation when short-term and long-term outcomes were evaluated [42].

Glutamine may also be used in powder form as part of an early enteral regimen in physiologic doses of 0.5 mg/kg/day in surgical and trauma patients. In patients with renal insufficiency, glutamine represents a nonprotein additional nitrogen burden and has been demonstrated to be harmful [38, 43]. In addition, there have been concerns that its anabolic effect could be permissive of tumor growth in patients with malignancy. Based on these observations, glutamine should only be given in physiologic doses, early in the post-injury course and withheld in patients with reduced kidney function or malignant tumors.

15.4 Metabolic Monitoring of the Catabolic Patient with an Open Abdomen

Careful metabolic monitoring results in more effective therapy. As critical illness is dynamic, caloric and protein requirements can change during its course. Calorie requirements based on resting energy expenditure should be rechecked weekly. Liberalize caloric support with prolonged illnesses to account for accrued deficits. Changes in protein turnover can be monitored with nitrogen balance studies weekly in those patients with adequate kidney function and urine output. Measurement of serum protein in the early phases of critical illness is not indicated, as they reflect the inflammatory milieu more than the state of nutritional adequacy [17]. However, as the illness progresses late into the flow phase, prealbumin levels coupled with C-reactive protein values will help determine the adequacy of nutritional support and the return of a more anabolic state [44]. If the patient progresses to a persistent catabolic state despite source control, increased caloric and protein support may be needed.

A major barrier in the delivery of adequate enteral nutrition to patients with an open abdomen is the interruption of feeding for operative and interventional procedures. Monitoring should include a daily assessment of the nutrition support that was *actually received* by the patient with reasonable attempts to minimize calorie and protein deficits. A "therapy bundle" is necessary to overcome the usual barriers of late initiation and loss of support due to frequent interruptions [17] (Table 15.6). Volume-based rather than rate-based ordering systems have demonstrated more effective delivery of calories and protein [45]. The volume of feedings required can be given over a cyclic period after feedings are resumed so that the prescribed nutrition is delivered. Calorie and protein deficits should be assessed on a daily basis to limit the underfeeding that often occurs.

Patients on parenteral nutrition should be monitored for return of gastrointestinal function and their ability to transition to enteral support. An important parameter to follow is adequate wound healing and the development of a robust granulation bed [44]. Keep in mind that inadequate wound healing could also be due to micronutrient deficiencies.

In all patients, routine electrolytes should be monitored and replaced, with a focus on magnesium and phosphorus. While hypomagnesemia may be associated with cardiac dysrhythmias, hypophosphatemia is common in patients with nutritional deficits and is associated with decreased respiratory muscle function and weaning failure.

15.5 Enteral Access Considerations

In patients without other options, feeding into the stomach can be attempted. The difficulty with this approach is that gastric ileus is often present in patients with an open abdomen. Patients must be carefully selected and monitored closely for tolerance in an attempt to avoid aspiration [17]. For this reason, current recommendations are to feed continuously or cyclically in a post-pyloric location in open abdomen patients [17]. This route can deliver adequate enteral nutrition more effectively and has been shown to be associated with a 30% decrease in the rate of pneumonia in enterally fed patients [46]. This type of access is technically difficult, but with expertise, can be achieved at the bedside. Fluoroscopy or endoscopy may be required in some cases.

As all of these patients are at high nutritional risk, there should be plans made for enteral access at operative procedures. The stability of the abdominal wall must be considered as enteral access can cause fistulae when misplaced or placed too close to the fascial edge (Fig. 15.1, gastric fistula). At the time of follow-up surgery or closure, suturing the stomach to the abdominal wall (Stamm technique) with radiopaque markers can be placed in anticipation for peg placement later when the patient is more stable and anabolic [47]. CT scan of the abdomen can be useful in determining the feasibility of gastric access when the abdomen is open (Fig. 15.2).

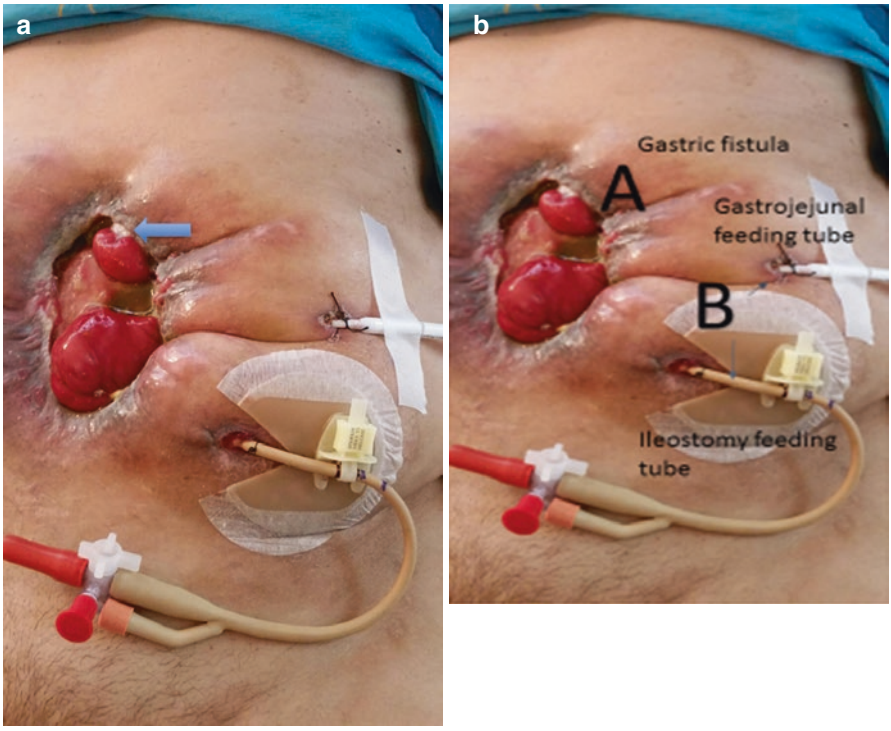

Fig. 15.1 (**a**) Open abdomen 10 months following car crash, patient referred with multiple fistulae. Note the *upper* gastric fistula which was caused by a gastrostomy tube placement via the skin flap close to the granulation plate. (**b**) Note fistuloclysis with gastrojejunal feeding tube via gastric fistula and ileostomy tube via small bowel fistula

Fig. 15.2 CT scan demonstrating the ability to determine safety for gastric access in open abdomen patient

15.6 Nutritional Support of the Enterocutaneous and Enteroatmospheric Fistula Patient

Enteroatmospheric fistulae are often present in conjunction with the open abdomen, as the result of failed intestinal anastomosis resulting in the open abdomen or the development of enteroatmospheric fistulae as a late complication. Fistulae deep in the peritoneal cavity cause peritonitis, and establishing adequate external drainage is more difficult. Fistulae through or adjacent to a granulated fused visceral block are also problematic [23]. Upon the development of this complication, patients should be started early on parenteral nutrition. There is no need to delay the institution of parenteral nutrition, as it has been reported that 76% of patients will require combination therapy for this illness [48]. Reoperative therapy for this complication should be put off for 6–18 months as injury to bowel during these procedures can convert a patient with Type II intestinal failure to Type III [49]. As soon as wound management is possible and drainage is adequate, patients should be fed proximally at a trickle rate to start. The early initiation of enteral feedings in open abdomen patients with fistulae (within 14 days) is associated with decreased mortality [50]. Intestinal mapping can be done to give the feasibility of success of proximal feeding and to establish availability of efferent limb for feeding [23]. The efferent limb of the fistula should be evaluated to exclude any leak or obstruction, so that enteral feeding can be started distally via "fistuloclysis" with elemental predigested formulas or fistula effluent [51] (see fistuloclysis access in Fig. 15.1).

In addition to aggressive calorie and protein support, support these patients with high doses of minerals and vitamins to replace losses. Zinc, vitamin C, copper, folic acid, and B12 should be monitored and replaced [23] (Table 15.7). Nutritional supplements that support wound healing with glutamine and arginine can be utilized as long as the patient is not in a state of severe sepsis. In addition, pharmacologic adjuncts are often helpful and include antimotility agents, antisecretory agents, bulking agents, and digestive supplements [23]. Octreotide should not be utilized except for short-term indications when output must be controlled as with skin grafting. Because it significantly decreases splanchnic and portal blood flow, it can inhibit intestinal rehabilitation [52] (Fig. 15.3).

15.7 Choosing an Enteral Formula

Whey-based protein formulations are higher in leucine and thus may have an anabolic advantage over soy- or casein-based formulas. Enteral formulas with 1.5 kcal/mL result in significantly more calories being delivered in critically ill enterally fed patients, without the adverse effects of increased gastric residual volumes or diarrhea [34, 53]. Some of these calorically dense formulas, however, may not have adequate protein, so that protein modules are necessary to meet nitrogen requirements. These can be given even before caloric goals are met to reach protein goals earlier. Because most formulas only contain 65% water, it may be necessary to administer hypotonic enteral fluid boluses in patients without intravenous maintenance fluid to avoid dehydration.

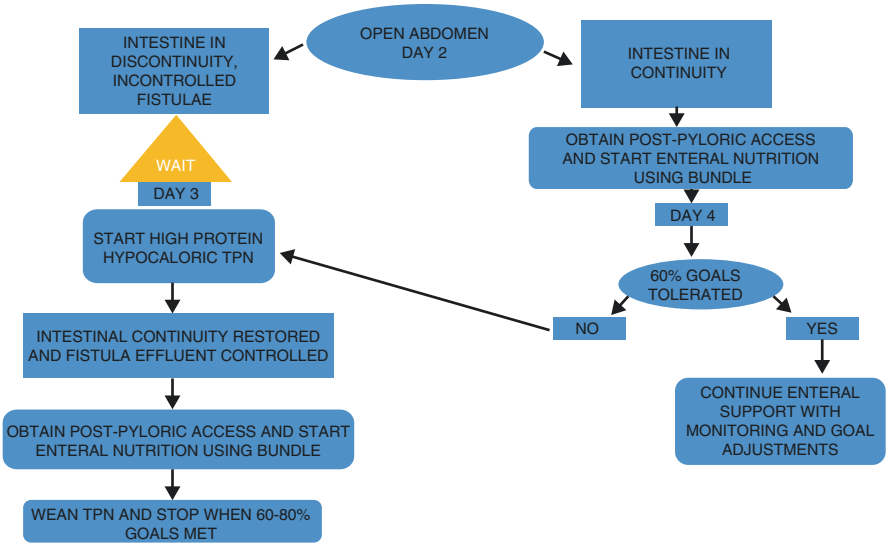
OPEN ABDOMEN
DAY 2
INTESTINE IN DISCONTINUITY, INCONTROLLED FISTULAE
INTESTINE IN CONTINUITY
WAIT
DAY 3
OBTAIN POST-PYLORIC ACCESS AND START ENTERAL NUTRITION USING BUNDLE
DAY 4
START HIGH PROTEIN HYPOCALORIC TPN
60% GOALS TOLERATED
NO
YES
INTESTINAL CONTINUITY RESTORED AND FISTULA EFFLUENT CONTROLLED
CONTINUE ENTERAL SUPPORT WITH MONITORING AND GOAL ADJUSTMENTS
OBTAIN POST-PYLORIC ACCESS AND START ENTERAL NUTRITION USING BUNDLE
WEAN TPN AND STOP WHEN 60-80% GOALS MET

Fig. 15.3 Open abdomen. Nutritional support algorithm

Concentrated formulas with more than 1 kcal/mL usually accomplish this goal by increasing fat calories. Do not routinely use formulas with 2 kcal/mL in critically ill patients, as these formulas have high fat content and may promote an inflammatory response [17]. However, recent animal studies have demonstrated that formulas with more fat calories may mitigate intestinal mucosal injury caused by the air exposure of the open abdomen [27]. Although volume losses are the common scenario in the early phases of the open abdomen course, as the patient becomes more chronic, volume restrictions due to underlying illness may become important. These calorically and protein-dense formulas allow adequate nutritional support in patients with these volume restrictions [17].

Patients with intestinal mucosal atrophy, those fed post-pylorically, and patients with gastrointestinal fistulae may require semi-elemental or peptide formulations. The use of immune-modulating formulas with arginine, glutamine, and nucleic acids has been controversial; however, they may be considered for early use in patients without sepsis [17]. There are also commercial products that have increased fiber and fat calories for diabetics, formulas that have adjusted electrolyte composition and concentrated calories for renal failure, and those with amino acid and protein adjustments for hepatic insufficiency. These products are far more expensive than the standard enteral products and should not be used without clear indications.

Administer a *soluble* fiber product, such as apple pectin or guar gum, routinely to all patients with a colon in continuity. Soluble fiber is a source of nutrition for the colonocytes and helps maintain gut barrier function. In addition, soluble fiber helps modulate and maintain a healthy colonic microbiome and thus helps to decrease the number of pathogens that may be found in the colons of critically ill patients. The recommended dose is up to 2.5–5 g every 6 h [17].

15.8 Complications

The most common complication associated with the enteral route of feeding is malnutrition due to serious underfeeding as a consequence of poor protocols and nursing techniques [54]. Aspiration is another serious complication of enteral feeding. The risk can be mitigated by keeping the head of the bed elevated at 30 degrees whenever possible. In patients demonstrating poor gastric emptying, post-pyloric access should be considered. However, whenever there is abdominal distention and evidence of severe feeding intolerance, tube feeding should be immediately decreased or discontinued with the concern for nonocclusive intestinal necrosis. Prompt recognition and surgical intervention for source control are needed for salvage from this devastating and highly lethal complication [55].

There are multiple complications that can occur with enteral access. Naso-enteral tubes can be incorrectly placed into the airway or become dislodged. Dislodgement of percutaneous tubes can result in gastrointestinal contents or tube feeding leaking into the peritoneal cavity. Finally, tube occlusion contributes greatly to underfeeding and can be avoided with frequent flushing and appropriate nursing care of feeding access tubes.

While underfeeding is the most common complication of enteral support, overfeeding is often a complication of parenteral nutrition and has been demonstrated to contribute to adverse outcomes. Overfeeding is associated with increased levels of metabolic stress and is permissive of hepatic steatosis. Associated hyperglycemia is associated with glycosuria, electrolyte derangements, hyperosmolarity, and an increased incidence of infections. This should be treated aggressively by adjusting carbohydrate calories and regulating insulin infusions to maintain blood sugars under 180 mg/dL [56]. Although insulin may be added to the parenteral nutrition formula at 50–60% of the previous day's requirements, this should only be done in stable patients who are not in flux with insulin resistance and should only cover the caloric load delivered in the parenteral formula. Hypercarbic respiratory failure may be induced by carbon dioxide production from excessive carbohydrate caloric loads. If indirect calorimetry is utilized, the carbohydrate load should be limited to keep the respiratory quotient <1. If indirect calorimetry is not available, the carbohydrate load should be kept to ≤4 mg/kg/min.

To decrease the risk of catheter-related sepsis in patients receiving parenteral nutrition, a dedicated access should be utilized for the parenteral nutritional products with protection from mixed use. Protocols utilizing alcohol and antibiotic locks can also be used as preventative measures.

Take-Home Box

- Start enteral feedings in all open abdomens with intestinal continuity as soon as they are hemodynamically resuscitated.
- Start TPN if at least 60% of nutritional requirements are not met by day 4.
- Use an enteral therapy bundle and volume-based ordering to maximize success with enteral feeding.

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The Nursing Management of Open Abdomen Patients

16

Rosemary Koehl Lee

16.1 Introduction

Florence Nightingale identified that the nurse's role is to put the patient in the best possible condition for nature to act [1]. The goal with the open abdomen patient is to put them in the best possible condition in order to close the abdomen as soon as feasible. The responsibility of the acute and critical care nurse is to provide safe passage for patients and their families. Providing "safe passage" is a cornerstone for nursing competencies based on the American Association of Critical Care Nurses (AACN) Synergy Model [2]. The purpose of this chapter is to convey the nursing management of the adult patient with an open abdomen (OA) so the patient and family can progress to health and well-being.

16.2 Patient and Family Preparation

The patient requiring an open abdomen may be one that presented to the emergency department and was urgently taken to surgery with minimal to no prep due to the life-threatening situation. Alternately, the patient may have been monitored and managed in the ICU for a medical cause of intra-abdominal hypertension (IAH), until the interventions failed and abdominal compartment syndrome (ACS) ensued and an open abdomen required.

Waking up from anesthesia with an open abdomen may be frightening for the patient and family. Letting them know what to anticipate will decrease the fear. Initially, the physician will inform them of the surgical plan, and the nurse may reinforce the explanations. The Abdominal Compartment Society has excellent

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information for patients on their website (http://www.wsacs.org/for-patients.html) [3]. Below are some frequently asked patient questions and answers obtained from the website:

Q. What is an "open abdomen?"
A. An operation where your doctor makes a large incision (cut) in your abdomen (belly) and leaves this incision open to relieve the high pressure that is preventing your organs from working properly. You will need another operation (or sometimes many) to close this incision when you are no longer sick. While your abdomen is "open" in the hospital, you will have a special covering to protect your organs. This operation is also known as "abdominal decompression."
Q. Why is an open abdomen necessary?
A. Organ failure, such as your heart, lungs, or kidneys not working properly, is the most common cause of death in the intensive care unit (ICU). Your doctors and nurses will do everything possible to prevent your organs from failing. If all other therapies fail, however, opening your abdomen may be the only way to save your life.
Q. How can I tell whether my abdomen is "open" or "closed"?
A. To close an open abdomen, your surgeon will sew both the skin AND muscles of your abdominal wall back together. Your abdomen is considered "closed" when BOTH layers of the abdomen have been repaired. You may, however, be too sick to close both layers at the same time. If this is so, your surgeon may sew only your skin together (called a "skin-only" closure) leaving your muscles apart to keep the pressure in your abdomen low. If this is not possible, your surgeon will take a piece of your skin (usually from the thigh) and place this over your organs (called a "skin graft" closure). In either case, you will have a hernia (a large bulge in your belly) until your surgeon feels you are ready to have another operation to close the muscle layer as well. While you have a hernia, you will need to follow the activity restrictions given to you by your surgeon. You may also need to wear an abdominal "binder" or "belt" to support your abdominal wall. Until both the skin and muscles of your abdominal wall have been sewn together, your doctors will consider your abdomen as being "open".
Q. Why do I have an open abdomen?
A. Because you are/were so sick, your doctor had to open your abdomen to save your life and prevent organ failure. In most patients, an open abdomen is temporary and your abdomen should be able to be closed in the near future.

Ideally having a patient/family conference with the health-care team preoperatively will provide the opportunity for questions to be asked and answered.

Knowing what type of temporary abdominal closure (TAC) the surgeon will use is important for the nurse to know. Please refer to Chap. 10 for the review of the various types of TACs and their pros and cons. The surgeon should prepare the health-care team on what type of TAC will be used, as the decision to leave the abdomen open is usually made preoperatively. Most TAC's need to be changed every 48–72 h by the surgeon.

16.3 Postoperative Management in the ICU

Immediate postoperative recovery of the open abdomen patient is similar to any major abdominal surgery, with a few exceptions. The standard practices in all major surgery patients in the ICU would be to maintain hemodynamic stability, normothermia, acid–base balance, hemostasis, and pain control. The patient most likely will be supported on mechanical ventilation, with lung-protective strategies [4]. There may be multiple surgical drains and tubes connected to suction or drainage devices. Patients with an open abdomen are still at risk for intra-abdominal hypertension (IAH) and require intra-abdominal pressure (IAP) monitoring.

Managing Hemodynamics Fluid loss is a major problem with the OA patient. In the first 24 h, the patient may require massive amounts of fluid and blood resuscitation in order to maintain hemodynamic stability [5]. The role of the nurse is to judiciously manage the IV fluids, blood products, and vasopressors while closely monitoring the hemodynamic parameters and urine output. The patient is also at risk for recurring abdominal compartment syndrome (ACS) and requires IAP monitoring.

There is evidence to indicate that [6] in the presence of IAH, static hemodynamic values, such as the central venous pressure (CVP) and the pulmonary capillary wedge pressure can be artificially elevated. Dynamic hemodynamic values such as stroke volume variation (SVV), pulse pressure variation (PPV), or global end-diastolic volume (GEDV) are better at predicting fluid responsiveness. Both SVV and PPV have limitations in their use. Assessing the changes in stroke volume (SV) or stroke volume index (SVI) using the passive leg raise has also been used to assess fluid responsiveness [7]. However, if IAH is present, the passive leg raise maneuver will be inaccurate [8]. Ensure the physician ordering the fluid resuscitation and vasopressors provides hemodynamic goals to be achieved and maintained. If using the arterial blood pressure as an endpoint of resuscitation, it is better to use the mean arterial pressure (MAP) than the systolic pressure [9].

Due to the substantial fluid losses, and the large amount of IV fluid intake, it is important to maintain an accurate intake and output (I&O). Fluid losses would be drained from the wound (surgical drains, negative pressure wound therapy), nasogastric tube, urine output, stool, fistula drainage (if present), and through diaphoresis. It is difficult to maintain an accurate I&O due to insensible losses like sweating and fistulae that are not bagged. It is recommended to also obtain daily weights of these patients to trend fluid retention [5].

Managing Intra-abdominal Hypertension (IAH) A hands-on abdominal assessment and measurement abdominal girth are not adequate indicators of IAH [10]. Trending IAP measurements are better guides to the development of IAH and ACS. A normal IAP in healthy subjects is 0–5 mm Hg; most critically ill patients have pressures between 5 and 7 mm Hg. Intra-abdominal hypertension is defined as a sustained or persistent elevation of the IAP of 12 mm Hg or greater. Abdominal compartment syndrome is defined as a sustained IAP of 20 mm Hg or

greater with new onset organ failure. Refer to Chap. 3 for a more in-depth discussion on how to properly measure IAP and what the significance of the readings is. Measure the IAP every 4 h and more frequently if active interventions are taking place to lower the IAP. The Abdominal Compartment Society recommends that critical care units develop a protocolized approach to measuring IAP and managing IAH.

The Abdominal Compartment Society has developed a medical management algorithm for the management of IAH (http://www.wsacs.org/images/algorithms/IAH_ACS_medical_management_2014.pdf.) [11]. From the medical algorithm nursing interventions can be gleaned. Many of these are basic nursing interventions that may get lost in the high-tech environment of critical care.

- Nasogastric (NG)/orogastric tube (OG):
  - Ensure the NG/OG is properly positioned in the lower half of the stomach by:
    Reviewing radiology reports.
    Aspirating of gastric contents.
    Note that insufflation of air and auscultating the left upper quadrant for sounds of air entry are not a recommended maneuver for tube placement. It gives false reassurance as the tube may be in the esophagus or lung and give the same sound [12].
- Ensure NG/OG tube is patent:
  - If patient has continuous or intermittent gastric suction, ensure the expected amount of aspirate is present. Maintain the suctions as ordered, and keep at the prescribed settings.
  - Irrigate the gastric tube as per unit policy. (See Fig. 16.1)

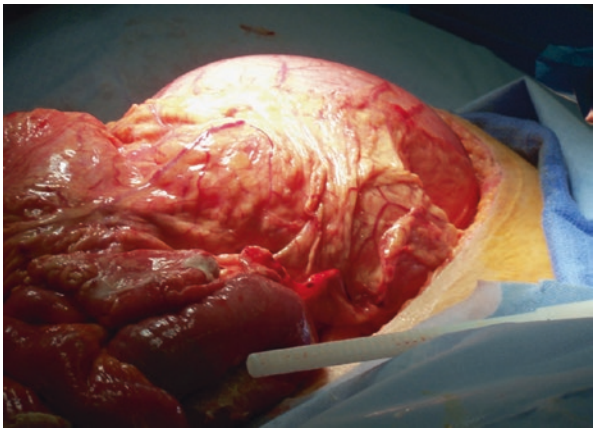

Fig. 16.1 This patient was being treated for peritonitis with an open abdomen and negative pressure wound therapy. Prior to going to surgery for this dressing change, the patient's IAP rose gradually from 12 to 20 mm Hg. Upon removal of the dressing in surgery, the grossly distended stomach was revealed. The patient's nasogastric tube was removed and found to be occluded. Upon placement of a new nasogastric tube, the stomach decompressed and IAPs returned to 10–12 mm Hg

Fig. 16.2 Abdominal CT scan showing bowel distended with stool

- Prevent constipation:
  - If the patient has an intact gut and is receiving enteral feedings, a bowel movement should be expected at least every 3 days or sooner.
  - Ensure patient has adequate enteral fluid intake to maintain stool consistency.
  - Review abdominal film and CT reports for the presence of stool or fecal impaction (see Fig. 16.2).
  - If not contraindicated, digitally inspect for a fecal impaction.
  - Most patients will be receiving opioids for pain which can cause constipation. Ensure that an escalating bowel regimen has been ordered:
    Stool softeners
    Laxatives
    Enemas

Nutrition Support Protein loss and malnutrition are problems of having an open abdomen. In order to promote wound healing, nutritional support is required. Depending on the patient's underlying condition either enteral (EN) or parenteral nutrition may be used. See Chap. 15 for a more in-depth discussion of nutritional support. This section on nutrition will concentrate on the nursing management of the enteral nutrition and will be based on the Society of Critical Care Medicine (SCCM) and the American Society for Parenteral and Enteral Nutrition (ASPEN) [13] guidelines for nutritional support in the critically ill.

Enteral support should not be initiated in the hemodynamically unstable patient. Patients with a MAP less than 50 mm Hg and on escalating doses of vasopressors should not receive EN. Gastric feedings can be given safely to the critically ill patient. If the patient is at high risk for aspiration a nasoduodenal or nasojejunal feeding tube may be used. Small bowel feeding tubes may also be required depending on the location of a bowel injury or presence of a fistula.

- EN should be started as early as possible within 24–48 h. It is important to collaborate with a dietitian or nutrition support team to identify the best feeding formula.
- Advancing the feeding to goal rate should be accomplished as quickly as possible within 24–48 h.

- Do not use gastric residual volumes (GRV) as part of a regular nursing routine. GRV's are not evidence based and do not correlate with feeding tolerance. It is better to asses for signs of feeding intolerance.
  - If GRVs are measured, it is recommended to not modify or stop the feeding until the GRV is greater than 500 mL.
- Monitor the patient for signs of feeding intolerance:
  - Vomiting
  - Diarrhea
  - Abdominal distention (increased IAP)
  - Complaints of discomfort
  - Decreased flatus
  - Decreased bowel movements
  - Greater than 500 mL GRV
- Minimize interruptions of EN feeds. Less than 50% of ICU patients reach and maintain their target feeding goal. This has been found primarily due to nursing care.
- Examples are:
  - Stopping or decreasing feedings for GRVs less than 500 mL.
  - Stopping feedings for nursing care, medical/surgical procedures or patient transport
  - Stopping feedings due to enteral medication requirements

It has been proposed [14, 15] that enteral feeds be prescribed as a daily goal volume rather than as an hourly goal rate. This proposal gives the nurse the option to temporarily increase the rate to meet the 24 h volume goal.

Mobility Early mobility for postoperative recovery has been a mainstay since World War II. Nurses are aware of the benefits of mobility to all body systems. In the acute postoperative phase of the open abdomen, the patient will require ventilator support, sedation, and analgesia requiring the patient to be bedbound. Despite hemodynamic instability, the patient may still be turned from side to side at least every 2 h. It is recommended to use the right lateral position first as this is better tolerated hemodynamically [16]. The lateral turns should be performed slowly to allow the baroreceptors to equilibrate.

In order to minimize IAH, it is recommended to elevate the head of the bed (HOB) no greater than 20 degrees. There has been research on the effects of various body positions on IAH; unfortunately the best position is supine with the HOB flat. Any bending of the torso will increase IAP readings [17]. This conundrum requires the nurse to closely assess the patient's response to position changes. Patients tend to stabilize within 5–10 min of a position change.

Once the patient has recovered from the acute phase and the temporary abdominal (TAC) closure is in place, it is important to initiate more aggressive mobility. The ABCDEF bundle provides guidelines on how to do this safely (see Table 16.1).

Table 16.1 Components of the ABCDEF bundle (http://www.iculiberation.org) [18]

Bundle	Component
A	Assess, prevent, and manage pain
B	Both spontaneous awakening trials (SAT) and spontaneous breathing trials (SBT)
C	Choice of analgesia and sedation
D	Delirium: assess, prevent, and manage
E	Early mobility and exercise
F	Family engagement and empowerment

Integument The goal of the TAC is to contain and protect the abdominal contents and minimize fluid and protein loss. It is the surgeon's responsibility to change the TAC, and it is the nurse's responsibility to maintain it. One of the challenges is to protect the skin from the drainage of the abdominal contents or the effluent from a fistula. These fluids are caustic to the skin which cause pain and delayed healing. Consulting a nurse specializing in wound care can assist the direct care nurse in developing ingenious ways of trapping and bagging wound and fistula effluent [19]. This aids in patient comfort and ease as well as decreasing the family's concern of the cleanliness of the patient environment.

Psychosocial/Financial Having an open abdomen and its sequelae of multiple surgeries, ventral hernia, wound drainage, etc., can have a debilitating effect on the patient and the family unit. Although most open abdomens can be closed within a week, others may take longer such as 6 months to a year. The long-term open abdomen patient may be discharged with the abdomen closed with the abdominal skin; however, the muscle and fascia are open underneath. This results in a large, unsightly abdominal hernia that will be repaired at a future date. In a study looking at the quality of life for these long-term patients [20], there was a high degree of depression (65%) and a concerning number with posttraumatic stress disorder (PTSD) (22.5%), and 42% did not return to work. In a study by Clark [21] the effects of insurance and race were studied as to their effects on outcomes of patients with an open abdomen. Race did not have a significant effect on patient outcomes; however, "self-pay" status had significant findings with an increased mortality rate. Knowing this, it would behoove the nurse to get the social worker/case manager involved early to deal with the anticipated financial and social issues upon discharge. The patient should be screened for depression and PTSD prior to discharge, so the proper therapy and pharmacological support may be started.

16.4 Summary

The purpose of this chapter was to present the unique nursing issues of the patient with an open abdomen. Along with the rest of the chapters in this book, it is hoped the nurse has been given the knowledge to put the patient in the best possible condition for nature to act and to provide safe passage to patients and their families.

Take-Home Messages

- Patients with an open abdomen are still at risk for IAH and need their IAP measured regularly.
- Basic nursing interventions can assist in minimizing IAH.
- Obtain goals to manage hemodynamics with fluids and vasopressors.
- Dynamic volume indicators are better than static hemodynamic parameters to assess fluid responsiveness.
- Maintain adequacy of enteral nutrition by using evidence based principles.
- Mobility is an important recovery intervention and begins with proper positioning.
- The patient should be screened for depression and PTSD risk.

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Open Abdomen Complications: Prevention and Management

17

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Highlights

- The advantages of open abdomen are not disputable, but the exposure of the abdominal viscera to the outer environment brings itself a great burden of dreadful complications.
- Open abdomen could be double-edged sword: prevention is still the best way to treat open abdomen complications.
- Open abdomen complications could be divided in medical and surgical.
- A multidisciplinary team formed by surgeons, anesthetists and ICU-attending personnel, nutritionist, psychologist, and specialized nurses with a specific expertise in open abdomen management is mandatory.
- Every attempt should be made to close the abdomen as soon as reasonably possible: a primary fascial closure within the first week of open abdomen seems to be the best way to reduce the complication rate.

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17.1 Introduction

The idea that leaving the abdomen open after an emergent laparotomy could be beneficial for patient survival dates back to the first years of the twentieth century, when Pringle originally described the term open abdomen (OA) [1] and in 1940 Ogilvie provided the first description of a temporary abdominal closure system for OA management [2]. Unfortunately, these techniques were associated with tremendous infective and hemorrhagic complications and were not routinely used until the 1990s, when the huge advancements in intensive care medicine and antibiotic therapy set the base for the development of the so-called damage control surgery (DCS). In fact, in 1983 Stone et al. [3] described the use of an abbreviated laparotomy in case of major coagulopathy, and in 1993 Rotondo et al. [4] borrowed the term damage control from the US Army to describe the abbreviated laparotomy as the only way to stop the lethal triad of hypothermia, coagulopathy, and acidosis. Furthermore, the increasing comprehension of the pathophysiology of trauma and abdominal compartment syndrome (ACS) made the open abdomen (OA) a cornerstone step of damage control surgery (DCS) [5].

Nowadays the advantages of OA in trauma patients are not disputable, but the exposure of the abdominal viscera to the outer environment brings itself a great burden of dreadful complications, such as malnutrition and starvation, because of the massive fluid and protein losses, fascial retraction and subsequent loss of abdominal domain, and the development of huge ventral hernia. The most feared and devastating complication is the formation of an enteroatmospheric fistula, which is associated with significant morbidity and mortality and characterized by extremely challenging critical care and nutritional management issues. EAF requires a multidisciplinary management and, despite the continuous progresses in OA management, EAF's mortality can nowadays still be as high as up to 40% [6, 7].

For all these reasons, surgeons must be aware that OA could be double-edged sword: prevention is still the best way to treat open abdomen complications.

17.2 Open Abdomen: Definition, Indications, and Temporary Abdominal Closure

The term open abdomen (OA) means not closing the abdominal fascia after a laparotomy and temporarily closing the abdomen with a temporary abdominal closure (TAC) technique. Currently accepted indications for OA [8–10] are: as part of DCS for trauma, intra-abdominal hypertension or abdominal compartmental syndrome (ACS) not responsive to medical therapy, acute mesenteric ischemia necessitating a second look, and impossibility to safely close the abdomen, either because of massive visceral edema and retroperitoneal swelling or because of the loss of abdominal wall (traumatic or after necrotizing fascitis). Further indications are infected pancreatic necrosis and abdominal sepsis, even if OA in these settings is still a matter of debate and comparative or randomized studies are lacking.

The role of the TAC is crucial, and some of the OA complications depend directly from the TAC method; theoretically, the ideal TAC technique is the one that:

- Acts like a barrier, thus completely isolating the abdomen content from the outer atmosphere
- Is not traumatic on the abdominal viscera and prevents the formation of adhesions
- Allows abdominal fluid collection and quantification
- Is quick and easy to apply, allowing easy access to the peritoneal cavity for further re-explorations
- Easily allows nursing maneuvers
- Prevents fascial retraction and protects the fascia for an eventual primary closure
- Is cost-effective

Surgical techniques for management of OA have evolved tremendously in the past three decades [11, 12], and currently the use of negative pressure wound therapy (NPWT) is the best management option [10, 13], especially in the setting of a septic open abdomen, and can be associated to different kinds of fascial traction systems [14–16].

17.3 Open Abdomen Complications

OA complications could be artificially divided in medical and surgical, where medical complications derive from the pathophysiology of a massive open abdominal wound and surgical complications arise from surgical maneuvers performed into the open abdomen.

Medical complications are:

- Dehydration, electrolyte imbalance, and acid–base disturbances
- Malnutrition and starvation

Surgical complications are:

- Bleeding
- Enteroatmospheric fistula
- Frozen abdomen
- Huge ventral hernia due to fascial retraction with loss of abdominal domain

17.3.1 Dehydration, Electrolyte Imbalance, and Acid–Base Disturbances

The presence of an open peritoneal cavity leads to massive fluid losses, both from direct secretion and from augmented evaporation. Furthermore, the patient with an open abdomen is usually a critically ill patient, where any imbalance of the

physiological status can be detrimental and where the presence of an organ dysfunction is extremely common. In this setting, it is mandatory to limit and to promptly correct any fluid loss.

Prevention In the beginning of the open abdomen era, the Bogota bag and the skin-only closure were the most used TAC [17, 18]; these techniques had the main drawback of fluid collection, because they did not allow the quantification of fluid losses and let most of the peritoneal fluid pooling into the OA. The best way to prevent dehydration and electrolyte imbalance is the utilization of a negative pressure TAC that allows a correct quantification of fluid losses and completely isolates the open abdomen, significantly reducing the excessive evaporation across the open wound [19].

Management Prompt fluid replacement should be meticulously balanced on the base of patient's fluid balance; volume status monitoring must be meticulous throughout the entire length of hospital stay; electrolyte and acid–base imbalance should be punctually corrected.

17.3.2 Malnutrition and Starvation

The importance of a correct nutrition in critically ill patients is well known since 1964, when Chapman et al. [20] noted that malnutrition was the leading cause of death in patients with enterocutaneous fistula and mortality was considerably reduced with a daily intake of at least 1500 Kcal. Patients with OA are continuously in a hypercatabolic state, due to the high amount of nitrogen losses from abdominal fluid and to the continuous activation of an exaggerated systemic inflammatory response, promoted by the secretion of pro-inflammatory cytokines in the open abdominal wound and by the eventual ongoing peritonitis. At the same time, the endless healing process consumes calories and proteins. This particular combination of factors explains the high risk of malnourishment and starvation typical of this population of patients; furthermore, in the unlucky event of EAF formation, the fluid and nutrient losses increase dramatically as well as the pro-inflammatory stimulus [21–23].

Prevention The creation of a personalized nutritional plan is of critical importance and is based on a correct nutritional assessment at the beginning of the OA treatment and the continuous monitoring of the nutritional status. Different empiric nutritional estimators are currently available (e.g., the Harris–Benedict equation) for a preliminary evaluation of the basal energy expenditure; subsequently corrections should be made in accordance to the specific characteristics of the single patient.

Management The first critical step is the establishment of a safe and effective feeding access, which could be either enteral or parenteral. An effective total parenteral nutrition (TPN) usually necessitates of a central venous access that carries

a numerous set of complication, either infectious, thrombotic, or related to the insertion site. Whenever possible, enteral nutrition (EN) is recognized as the best way for prolonged feeding, and the benefits of early EN compared to TPN are indisputable [19, 24–33] and include preservation of the intestinal mucosal barrier and its immunological function [27] and a reduced rate of infectious complications, without affecting fascial closure rates even in patients with OA and EAF [26, 28]. EN access should be easy to establish and maintain and should be tailored upon specific patient characteristics; multiple different possibilities are available and include nasogastric or nasojejunal tubes, jejunostomy, and fistuloclysis in the presence of an EAF [31, 34]. EN should be started as soon as possible, with the added aim of manipulating the stress response to injury and infection and in order to improve the patient outcome [35].

Furthermore, supplementation of vitamins and trace elements is of utmost importance, addition of 2 g of nitrogen per liter of abdominal fluid lost from the OA is necessary to maintain a positive nitrogen balance [33], and patients with high-output EAF require 1.5–2 times the usual calories.

The application of a NPWT dressing and the subsequent promotion of peritoneal fluid egress could slow the catabolic process by reducing the local concentration of pro-inflammatory mediators [36].

17.3.3 Bleeding

Theoretically, the creation of a negative pressure inside the open abdomen could increase the risk of hemorrhage.

Prevention If active bleeding or coagulopathy is suspected, the optimal amount of negative pressure should be decreased from the ideal −125 mm Hg to a safer value of 75 mm Hg for the first 48 h [10, 37].

17.3.4 Enteroatmospheric Fistula

An enteroatmospheric fistula (EAF) is an opening in the GI wall occurring in the setting of an open abdomen, creating a communication between the GI lumen and the external atmosphere. EAF has exclusive features, i.e. the absence of a proper fistula tract and the lack of well-vascularized surrounding tissue, which make its spontaneous closure extremely unlikely. Additionally, the spillage of enteric content directly into the open peritoneal cavity leads to a continuous peritonitis, accompanied by its harmful effects.

EAF incidence rates are extremely variable between different studies and this variability could be related to the multiple possible etiologies of EAF and to the different indications for OA. It is recognized that a septic open abdomen is more fistula prone than a non-septic one: in a recent systematic review, Bruhin et al. [38] confirmed this hypothesis and found a rate of EAF of 12.1% in septic OA, compared to

a 3.1% of the non-septic OA. Atema et al. [39], in their meta-analysis, reported rates of fistula ranging from 5.7 to 17.2% in non-trauma patients, while the incidence of EAF in OA for abdominal sepsis could reach values as high as 54.5% [40].

Risk factors for EAF development are still mainly undetermined, but traumatic insults to the bowel certainly play a pivotal role; this is indirectly demonstrated by the evidence that the incidence of EAF increases with the increase in duration of OA [41] and, even more interestingly, EAF occurrence shows a direct correlation with the number of surgical re-explorations [42, 43]. A recent multicenter prospective observational study by Bradley et al. [44] confirmed these findings and demonstrated that large bowel resection, large volume resuscitation, and an increasing number of re-explorations were predictors of the development of EAF in trauma patients. Other conditions that could lead to fistula formation are anastomotic leakage, ongoing bowel ischemia, distal bowel obstruction, and adhesion of the bowel to the fascia that may cause an EAF.

Prevention The best way to treat an EAF is to prevent it. Different factors, which are known to be involved in EAF development and maintenance, should be identified-treated promptly and prevented whenever possible; these factors are traditionally listed in the mnemonic acronym *FRIENDS* (*F* Foreign body; *R* Radiation; *I* Infection or Inflammatory bowel disease; *E* Epithelialization of fistula tract; *N* Neoplasm; *D* Distal obstruction; *S* Short tract <2 cm).

Given that desiccation of bowel loops and unrecognized microtrauma seem to be the most important causes of fistula formation, any possible action that could prevent them must be put into effect: (1) any rough and/or direct contact between the viscera and the devices used for temporary abdominal closure must be avoided or minimized; (2) nonabsorbable prosthetic meshes are not recommended, as they can erode the abdominal viscera leading to fistula formation; (3) make sure that TAC is completely occlusive and prevent bowel loop exposure and desiccation; (4) close the abdomen as soon as possible; (5) carefully plan dressing changes; (6) dressing changes should be performed by senior attending surgeons with specific expertise in OA management and, possibly, every time by the same team ("same team concept") [45]; and (7) if fascial closure is precluded, early split-thickness skin graft or cadaveric skin graft over the granulating viscera could help to protect the viscera.

Management The acronym *SNAP* (*S—sepsis and skin care*, *N—nutrition*, *A—anatomy, P—plan*) [46] condenses the key points of EAF management. Firstly, in the acute phase, *s*epsis control and *s*kin care (and wound care) are mandatory to stop the systemic derangements of the patient, driven by the severe peritonitis caused by the leakage of enteric content directly into the peritoneal cavity. The next critical step is the classification of the EAF, because different kinds of EAF require different approaches. The classification is strictly related to the exact determination of the *a*natomy of the EAF, and critical goals are (1) determination of exact location of the fistula, to allow a correct quantification of fluid and electrolyte losses (knowledge of the electrolytes composition of the GI fluids is necessary [30]), and (2) determination of the total length of the remaining bowel and the maximum length

of continuous surface of bowel available for absorption. *N*utritional support can be extremely challenging, and main principles are similar to what was previously said for OA patient, with the adjunct that EN can increase EAF output. Still, the only contraindication to EN is a usable bowel shorter than 75 cm [10, 30]. For this reason a reduction of fistula output could be tremendously helpful for a correct effluent control and H_2 receptor antagonists, proton pump inhibitors [47], somatostatin, and its analogues [27, 47–49] could be used to reduce intraluminal secretions. Nevertheless, a tendency toward caution in their use is advisable, because they can also inhibit the secretion of insulin and glucagon and reduce splanchnic blood flow.

For a proper surgical management of EAF, a well-conceived *p*lan, prepared by a multidisciplinary team that gathers together surgeons, anesthetists, nutritionists, nurses, wound experts, and stoma expert, is mandatory. The evident best therapeutic option, i.e. a proximal bowel diversion, is frequently not feasible for several reasons, such as mesentery retraction and edema, "frozen abdomen," and abdominal wall tissue loss or retraction. A myriad of different surgical techniques is described so far in literature [50–77], and none of them could fit every patient or every situation. Hypothetically, the best EAF management technique should achieve the sequent goals: (1) completely isolates fistula from the remaining open wound, (2) is as atraumatic as possible on the fistula itself and on the frail surrounding viscera, (3) allows fistula output collection and quantification, (4) is quick and easy to apply, and (5) is cheap and made of easy-to-find materials.

Di Saverio et al. recently proposed a clinical algorithm and an extensive review of the literature [78] and a new classification of EAF [79], with the aim to promote and stimulate a rationalization of the surgical approach to this dramatic complication. This algorithm reassumes the most commonly used techniques into an easy-to-read diagram, and we refer to these papers for a complete description of all the available surgical options for fistula diversion.

As said above, EAF unique features make its spontaneous closure extremely improbable, and the main goal of surgical management is promoting the EAF to become a chronic but well-controlled fistula. Every management option is therefore intended to be a bridge from the acute phase to a chronic phase, when sepsis and peritonitis are resolved and patient has recovered, is well nourished and in a physiological balance, and is fit for definitive surgery for fistula takedown and abdominal wall reconstruction. This condition usually requires at least 8–12 months; in this lapse, visceral adhesions are supposed to get loose enough to allow a correct and safe surgery.

17.3.5 Frozen Abdomen

The condition called frozen abdomen is the result of a prolonged OA and is characterized by the presence of dense adhesions among abdominal viscera and between bowel loops and abdominal wall; a continuous layer of granulation tissue overlays the guts fixing them into an inextricable single mass. This situation configures the Grade III in Bjork's classification of OA revised by World Society of the Abdominal

Compartment Syndrome [80]. In the presence of a frozen abdomen, every surgical maneuver is precluded, and great attention must be paid in dressing changes to prevent the dreadful formation of EAF, which is relatively frequent in this situation. The natural evolution of the OA with frozen abdomen is the formation of a huge ventral hernia.

Prevention Prevention is the key, because there is no going back from a frozen abdomen situation. Prevention is based mainly on the application of TAC methods that contrast fascial retraction (i.e., NPWT with or without fascial traction systems) and prevent adhesion formation, by reaching deep bilaterally in the paracolic gutters and completely isolating the abdomen from the outer environment. The use of instillation with NPWT seems to improve bowel loop moisture, preventing adhesions and improving abdominal closure rates [10, 81]. Every effort should be made to obtain an early fascial closure, because it is well known that the rate of OA complications increases dramatically after the first week of OA treatment [16, 41, 42, 82, 83].

Management Once the abdomen has become frozen, the only management possibility is to promote the granulation of the exposed bowel, to protect the viscera from mechanical trauma, and to limit the formation of further adhesions. The application of absorbable synthetic mesh or nonadherent silicon dressings together with NPWT can promote granulation over the viscera. Some animal models suggested that NPWT could be linked to EAF formation [84, 85]: this controversy has now largely been contradicted [13, 50, 86], and the clinical impact of these observations is uncertain, but still a tendency toward caution and toward use of lower pressures in suction [13] is advisable. After granulation, biological dressing (i.e., human acellular dermal matrix, cadaveric split-thickness skin graft, or autologous skin graft) [52–54] can create a further natural protection over the exposed bowel and accelerate the process of epithelialization. A stepwise and cautious attempt to fascial closure could make a worthwhile contribution to the reduction of the amplitude of the subsequent ventral hernia.

17.3.6 Huge Ventral Hernia

Traditionally, the abdominal closure after OA was obtained allowing the open wound to granulate, thus creating a *planned ventral hernia*. However, this approach is associated with an increased risk of OA complications and puts a great physical and psychological stress on the patient. A recent study from Zosa et al. compared long-term outcomes for patients with OA with primary fascial closure and planned ventral hernia and found a higher complication rate in the first year for the hernia group with a return to the normal life activity postponed of 1 year; even more interestingly, these authors found also an increased rate of hernia recurrence after planned ventral hernia and surgical delayed reconstruction compared to patients

where primary fascial closure was achieved (recurrence rate 18.8% versus 8.5%) [87]. Schmelzle et al. performed a retrospective study excluding all the patients with the OA being closed in the first 7 days and found an extremely low rate of primary fascial closure [43]. For these reasons, current tendency is toward a delayed primary fascial closure, i.e., the closure of the abdominal fascia, as early as possible during the first hospitalization.

Prevention In this context too, prevention is crucial. It is demonstrated that fascial retraction begins as early as day 3 [16]; hence it is important to choose a TAC that could reduce fascial retraction from the very beginning of OA. NPWT has demonstrated a better tendency toward primary fascial closure when compared to other TAC methods, especially if associated with fascial traction systems [14, 15]; a 2014 meta-analysis of the outcomes for the currently available TAC methods found an overall weighted fascial closure rate of 50.2%, with the highest fascial closure rate reported in series describing NPWT associated with fascial traction systems (closure rate 73%) [39]. Another recent meta-analysis found a significant reduction of mortality rate between OA patients treated with NPWT versus patients without NPWT (mortality 28.5 versus 41.4%) [45]. It is easy to understand that the earlier is the achievement of a safe primary fascial closure, the lower the complication rate became.

Management The management of these huge planned ventral hernias is extremely challenging and brings with itself a great burden of morbidity and mortality that should not be ignored. Anyhow, definitive surgical treatment should be performed only after the complete recovery of the patient from the initial insult; it requires an accurate planning and preoperative nutritional and physical patient preparation. Multiple approaches for definitive surgery may be required in a multistep fashion, and this goes beyond the intent of this chapter; we remand to the specific literature for the complete description of the available surgical options [72, 88–92].

Conclusions

Although open abdomen is a lifesaving procedure for many critically ill patients, it brings with itself a whole new set of dreadful complications, such as the enteroatmospheric fistulae. Prevention is the key point when the use of OA is necessary: the patient should be managed by a multidisciplinary team formed by surgeons, anesthetists and ICU-attending personnel, nutritionist, psychologist, and specialized nurses with a specific expertise, and this team must be aware of the OA-associated morbidity. Given the indisputable increase in morbidity and mortality caused by a prolonged OA, every attempt should be made to close the abdomen as soon as reasonably possible. Even if the choice of the right timing for abdominal closure is extremely hard to make and a safe primary closure could be precluded in some cases, a primary fascial closure within the first week of OA seems to be the best way to reduce the complication rate.

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Mistakes in the Open Abdomen

18

Michael Sugrue and Mary Connolly

18.1 Introduction

Over the last 20 years, outcomes in emergency surgery and trauma in patients with open abdomens have improved tremendously [1]. However mortality remains high with significant variation [2, 3]. Prevention of exsanguination and reduction of massive transfusion requirements, with earlier hemorrhage control and selective use of the damage control techniques, are the key advances in trauma. Increasingly, damage control and open abdomen (OA) are being explored in emergency surgery [4]. This along with a proactive approach to sepsis control in emergency surgery has ensured mortality rates of less than 10% in some series. As part of damage control, the open abdomen has been widely used with undeniably improved outcomes in trauma. The indications for the open abdomen are shown in Table 18.1. There are obvious differences in indications for an open abdomen (OA) between general surgery and trauma.

The use of an OA has not been without its controversies. When *Fietsam* first coined the term abdominal compartment syndrome (ACS) in 1987, there was an understandable reluctance to leave a patient with their abdomen open [5]. Problems with dressings, both in terms of maintaining an intact abdominal domain and managing fluid loss, confounded by a high fistula rate polarized views relating to the benefits of the OA (Fig. 18.1).

With innovation came enthusiasm, and some centers, particularly in the Southern USA, had a 25% OA rate in trauma. Recently, *Bruns* reported 28% of their emergency surgery population had an OA [6]. Some of the series reflect specialized practice and, in the case of *Bruns* and colleagues, reflect their access to a super tertiary referral practice.

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Table 18.1 Classification of errors in the open abdomen

Preoperative
– Resuscitation errors
– Failure to obtain sepsis control
– Delay or procrastination
Pre-incision planning
– OR/ICU systems for open abdomen (KPI and outcomes)
– Avoiding fistula
– Stoma planning
– Avoiding anastomosis
– Incision planning
Maintaining abdominal domain
– Dressing choice
– Dynamic closure techniques
Closure plan
– Written closure plan
– De-resuscitation protocols
Abdominal wall optimization place
– Preventing/reducing risk of dehiscence

Preoperative mistakes generally occur in the emergency department, usually in the form of excessive fluid resuscitation, delay in antibiotic therapy, vasopressor procrastination, and slow responsiveness to altering physiology markers in the severely ill patient, either from sepsis or trauma.

How often do you see large volumes of fluids being infused into a normotensive patient? While it has been suggested that the fluid restriction pendulum has swung too far, patients in general remain over-resuscitated [7]. It is however important that there is appropriate fluid resuscitation before surgery, especially where there is documented significant base deficits (>6 mmol/L), indicating hypoperfusion. Early critical care involvement is essential. A consultant (attending) surgeon needs to be present to support and direct the resident in their decision-making in the emergency department in these critically ill patients.

18.2 Mistake 1: Telephone Medicine Does Not Work

In the early phases of resuscitation, failure to institute a "crystalloid cap," whereby fluid resuscitation of more than 2 L, should be flagged. One should check the appropriateness of further fluids and assess the need for supplemental vasopressors.

18.3 Mistake 2: Failure to Institute a Crystalloid Cap

Failure to limit crystalloid use will increase tissue edema and ultimately mortality. In the trauma patient, early hemorrhage control (blunt trauma 45 min and penetrating 15 min) is essential, and in the septic hypotensive patient with peritonitis, source

control needs to be achieved in under 2 hours [8]. Delay to laparotomy will increase mortality in septic shock, irrespective of whether it is a perforated duodenal ulcer or diverticular perforation.

18.4 Mistake 3: Delay to Investigation and Definitive Care

18.4.1 Perioperative

A bundle approach to the patient who potentially needs an open abdomen may help, firstly avoiding the need to keep the abdomen open and secondly reducing complications [9]. In general, one should ask why the abdomen is *not* being closed. To leave an abdomen open is morbid and sets a complex care path for the patient. The absolute indications to leave the abdomen open are shown in Table 18.2. They are not that common.

The key to a good outcome is excellent surgery (loving your tissues) and keeping the patient adequately perfused in the post-operative setting. If closing the abdomen means progression of intra-abdominal hypertension (IAH) to full blown ACS, then clearly it was a mistake to close. Predicting those patients who will develop IAH is not difficult, but predicting those whose IAH will progress to ACS is more difficult. A prophylactic OA rate should be around 5% of all laparotomies, with perhaps 2% in non-trauma patients and about 8% in trauma. *Harvin* highlighted problems with the overuse of damage control surgery [10]. Damage control laparotomy (DCL) was associated with an 18% increase in hospital mortality, a 13% increase in ileus, a 7% increase in enteric suture line failure, an 11% increase in fascial dehiscence, and a 19% increase in superficial surgical site infection [10]. The increasing use of laparoscopic approaches to intra-abdominal sepsis will affect this rates in time.

Therapeutic open abdomen and decompression are still debated. It is currently not possible to predict impending ACS or identify those who will not benefit from aggressive medical strategy but instead require surgical decompression. Before planning surgery, your hospital will need a familiar, consistent dressing approach, with protocols for measuring IAP [11]. Stoma management needs to be thought of before placement, and one should avoid placing ostomies medially (Fig. 18.2).

Particular care should be taken before performing an anastomosis and leaving the abdomen open. The anastomotic leak rate will be over 20% if you have not closed the abdomen within 5 days. You are then not just faced with a leak but a leak in a semi-frozen abdomen.

Table 18.2 General indications for the open abdomen

Indications	%
Damage control (DC)	40
Facilitate early second look	25
Multiple	20
Overwhelming fecal contamination	10
Decompression to prevent treat ACS	5

18.5 Mistake 4: Performing an Anastomosis and Not Getting the Abdomen Closed in <5 Days

If an anastomosis is performed in the presence of a lot of tissue edema, then a hand-sewn technique, rather than stapled, is preferable. If a stapled technique is used, the stapler must be applied for 30 seconds before firing and the anastomosis kept as far away from the open abdomen as much as possible. In general, anastomoses should be avoided in an open abdomen.

A lateral stoma should be considered, to avoid potential leak with negative pressure wound therapy (NPWT) dressings. Remember a leak is a major mistake and may lead to evisceration. Feeding jejunostomies should be avoided, if possible, as they may leak due to the lack of adhesions from multiple repeat operations. Nasogastric (and rarely nasojejunal) feeding tubes are a much better options.

A transverse laparotomy, while possible, does not give the same access. Avoid tramlining incisions as they will result in abdominal wall necrosis (Fig. 18.3).

18.6 Mistake 5: Errors in Measuring IAP

The WSACS (World Congress of Abdominal Compartment Syndrome) recommends that IAP be measured every 4–6 hours in critically ill patients who demonstrate risk factors for the development of IAH or ACS. Once a nursing shift or eight hourly is probably adequate. Remember, intermittent IAP is, however, somewhat artificial and doesn't truly reflect the day-to-day activities of an ICU patient. Clinical examination is not a reliable measure of IAP. So get rid of your tape measure and stop drawing on the abdomen [12]. The standard for IAP measurement is the intravesical technique with instillation of 25 mL of saline. A Foley manometer U Tube is widely used in Europe and costs only about €25 per patient (Fig. 18.4). IAP measurements do not vary much, so an unexpectedly high IAP should prompt a check for a blocked catheter.

18.7 Mistake 6: Failure to Measure IAP in a Post-laparotomy or OA Patient

18.7.1 When to Decompress and Leave Abdomen Open

This is not always straightforward, but certainly it should be *decision before incision*. *Ivatury* was one of the first to advocate prophylactic decompression [13]. Prophylactic decompression should be considered in patients shown in Table 18.3.

Table 18.3 Definite indications for prophylactic open abdomen

• Mesenteric ischemia with or without anastomosis
• Unstable patients undergoing laparotomy for severe peritonitis
• Abdominal wall loss
• Avoiding polycompartment syndrome in massive resuscitations
• *Remember, your anesthetic team may not tell you that your patient is on high-dose vasopressors*

Patients with intestinal ischemia will benefit by leaving the abdomen open, not only to facilitate re-inspection and potential further resection but also to benefit from NPWT effects on the microvascular environment. NPWT reduces edema, reducing IAP thereby increasing perfusion. This is particularly important in venous mesenteric infarction. These patients do not need a long abdominal incisions as they have a resection that rarely gets tertiary ACS.

18.8 Mistake 7: Thinking You Should Never Decompress a Pancreatic Patient

A big challenge in clinical practice remains patients with severe pancreatitis, with IAPs >25 mm Hg, increasing abdominal signs and a worsening respiratory status. *Smit* recently highlighted the deleterious effect of IAH in pancreatic patients. Patients with high IAP and tissue edema may get intestinal ischemia [14]. This ischemia is multifactorial, but high pressure is a key factor, and ascitic fluid must be removed.

Mentula [15] had previously suggested early decompression, in the right patient, improves outcome in severe acute pancreatitis [15]. However, many leading pancreatic surgeons are vehemently opposed to opening the abdomen. Failure to open (anterior abdominal compartment) may result in mesenteric ischemia or delayed bowel resection for gangrene. Before decompression, one should consider percutaneous drainage of ascites if present and also complete paralysis.

18.9 Mistake 8: Carelessness at time of temporary and permanent closure of the Open Abdomen

It is a significant error to expect a junior resident to create the open abdomen dressing or close an OA. While the rectus contains "only" the inferior epigastric neurovascular bundle, failure to oversee the correct insertion of the protective sheet or suturing of the mesh, for mesh-mediated traction, may result in abdominal dehiscence. Dressing displacement may occur in ICU and can lead to the patient returning to the OR.

Patients at high risk of persistent or recurrent tertiary peritonitis and those with large-volume fluid resuscitation need a dynamic closure system. There are many different varieties, but currently, the best is mesh-mediated traction system, combined with negative pressure dressings. It is a mistake not to use this from the first operation [16]. The Bogota bag is reserved only for resource-challenged countries. Remember if you use a Bogota bag, it can split—just like a car windscreen. Using other such systems, including the Barker dressing, will increase the patient's mortality [17]. Particular attention to sterility is even more important when patients have an aortic graft or retroperitoneal necrosis. Suggestions to reduce adverse outcomes are shown in Table 18.4.

You should close EARLY. At the index operation for the open abdomen, the closure plan should be 24 hours for post-ischemia resection, 48 hours post-trauma packing, and 7 days for pancreatitis. Failure to achieve this will negate many of the

Table 18.4 Mistakes to avoid in the open abdomen

Mistake	Preventative strategy
Adhesion formation increasing the complexity of re-exploration	Careful insertion of plastic protective layer as far lateral as possible
Prevent lateralization of abdominal wall	Dynamic traction device
Prevent loss of fascia and skin	Love your tissues
Prevent ileus	Early enteral feeding Avoid opioids
Prevent development of EAF	Avoid anastomosis—but if you have to always hand sew Protect anastomosis with omentum
Prevent malnutrition and impaired healing	Close early Optimize nutrition
Reduce tertiary peritonitis	Bundle approach at first surgery
Early detection of recurrent sepsis	IAP/CRP trends and scan if in doubt

benefits of the OA and result in a high fistula rate and persistent catabolic state. However up to 15% need re-exploration due to tertiary and quaternary ACS or wound dehiscence. At re-exploration the abdomen needs to be washed out carefully with sequential tightening of the mesh to reduce lateralization, the enemy of final closure. Obtaining closure involves a team approach involving ICU, nursing, medical, and theater staff. *Miller* in a large series identified that if the abdomen was not closed within 7 days, the complication rate increased dramatically [18]. In *Atema's* review [19], when NPWT was used without fascial traction, a fistula rate of 14.6% was seen. But when NPWT was combined with continuous suture or mesh-mediated fascial traction, fistula risk dropped to 5.7%.

There will be a number of patients that you will not be able to close; if you have not closed by day 10, you will need to apply additional cover. A biologic mesh is probably preferable. Other options include skin coverage, which generally will require a releasing incision or a skin graft. The advantage of grafting is that it will correct the catabolic effect of the open abdomen quickly but will doom the patient to a delayed ventral hernia repair. A biological bridging mesh is probably most suitable. While *Cothren's* group reported up to a 100% fascial closure, a recent large series from Germany of 355 patients showed that even when the data is stratified by year, patients operated within the last 2 years of the study period (2011–2013) achieved only 49% fascial closure [20, 21].

To facilitate closure, a number of novel techniques have been proposed. One of the most promising is peritoneal resuscitation with hyperosmolar renal dialysis fluid [22]. Don't ask for this in the middle of your operation, as there are over 30 different bags of dialysis fluid; avoid the embarrassment and plan ahead! Hypertonic saline intravenously has been used to increase the fascial closure [10]. When closing, nearly 15% will dehisce, consider a supplemental prophylactic onlay mesh or even the retention sutures.

The skin should not be left open unless there is significant contamination. While there will invariably be contamination, overt infection is rare, due mainly to effective NPWT, and this will allow a subcuticular stitch. The wound appearance and complication rate will be reduced by the application of a closed incision negative pressure therapy system.

Table 18.5 Quality care monitors abdominal compartment syndrome

• Measurement of IAP post-emergency laparotomy patients
• Active ICU care plan to deal with ACS preventative strategy
• Documented closure plan in index open abdomen operation report
• Fistula rate <10%
• Use of NPWT abdominal dressing in open abdomen patient

The key to avoiding fistula formation is closing the abdomen early. Generally abdominal wall closure will have to be delayed where there is a fistula. Skin closure can be with subcuticular (remember love your tissues), even with existing colonization. If there is some purulent material in the wound, loose interrupted sutures can be used. If there is frank pus, a negative pressure superficial wound system will facilitate management, and skin edges will heal by secondary intention.

Conclusion

In conclusion, attention to fine detail is vital, with early presence of senior decision-makers in the emergency department and/or ICU, and prompt source and hemorrhage control will provide the best outcomes. Recently the World Society of Emergency Surgery in conjunction with the Abdominal Compartment Society and Donegal Clinical Research Academy recommended the adoption of new key performance indicators for the open abdomen (Table 18.5) [23, 24]. Errors may cost the patient their life and leave a stain on your conscience.

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Definitive Closure, Long-Term Results, and Management of Ventral Hernia

19

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Key Points

- Open abdomen often leads to giant abdominal wall defects.
- To prevent abdominal compartment syndrome and to allow tension-free closure, abdominal wall advancement is often needed.
- Often used advancement techniques are Rives–Stoppa procedure, anterior component separation (modified Ramirez technique), and posterior component separation technique (transversus abdominis release (TAR)).
- When advancement is not sufficient, additional measures such as bridging, pneumoperitoneum, and/or botulin toxin injections can be helpful.

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19.1 Introduction

Several clinical situations can lead to an open abdomen treatment period. The survival of conditions requiring open abdomen has greatly increased over the years [1]. In most cases, the first abdominal wall closure is temporary abdominal wall closure. However, in many cases, open abdomen treatment results in a remaining large ventral hernia. These large hernias are associated with complaints of severe back pain, ventilator function disturbances, and chronic wounds or enterocutaneous fistulae, resulting in a great decrease in quality of life [1–3].

The repair of large ventral hernias is technically challenging, and it is associated with long hospital stay, high rates of reoperations and readmissions, impaired wound healing, and high recurrence rates [4–8].

This chapter will describe the basic principles of ventral hernia repair after open abdomen treatment, the different treatment modalities, and future directions.

After open abdomen treatment, muscle tension and chronic lateral muscle retraction cause reduction of the intra-abdominal volume (loss of domain). This can cause problems after closure like severe postoperative pain, abdominal hypertension, wound dehiscence, ventilatory and/or pulmonary problems, and higher risk of recurrence [9].

In large ventral hernia repair, the loss of domain is an important principle. In a study evaluating volumetric measurements on CT imaging to predict tension-free closure, Sabbagh et al. established that a ratio of incisional hernia volume/peritoneal volume (IHV/PV) <20% is predictive of tension-free fascial closure. When IHV/PV is greater than 20%, only 12.5% of patients underwent a tension-free closure without resection [10]. This is supported by Kingsnorth et al. who hypothesized that considerable physiological adaptation (primarily respiratory) is necessary if the herniated volume exceeds 15–20% of the size of the abdominal cavity [11, 12]. After open abdomen, the loss of domain is often seen, increasing the chance of complications after closure surgery.

In addition to this, in 2014, Slater et al. defined criteria (Tables 19.1 and 19.2) based on clinical findings, to classify ventral hernias as complex [13]. With higher complexity, the number of perioperative measures, risk of complications, and costs rise. Because of the abovementioned reasons, patients with a history of open abdomen are almost always defined as complex ventral hernia patients. Therefore, the definitive closure after open abdomen requires specialized management and techniques. These will be discussed in this chapter.

19.2 Techniques for Abdominal Wall Domain Advancement

Already stated almost 60 years ago, tension-free mesh repair remains to be considered the gold standard for ventral hernia repair [14–16]. In patients with a history of open abdomen, tension-free closure is not possible without creating advancement of the abdominal wall domain in most cases. In the past decades, several techniques have been developed to gain advancement. The choice of different techniques

Table 19.1 Criteria for definition of a complex abdominal wall hernia defined by Slater et al.

Size and location	Large size, >10 cm width Parastomal, lumbar, lateral, or subcostal Loss of domain >20%
Contamination and soft tissue condition	Wound environment with surgical wound class III (contaminated) or IV (dirty) Full-thickness abdominal wall defects Loss of substance Distorted anatomy Denervated muscles Skin grafts Wound ulcers/non-healing wound Open abdomen Disease related (omphalocele, necrotizing fasciitis) Presence of enterocutaneous fistula
Patient history and risk factors	Recurrent hernia after earlier mesh repair or component separation Comorbidities/factors impairing wound healing: obesity, diabetes, old age, steroid use, poor nutritional state Increased intra-abdominal pressure: obesity, COPD Previous wound dehiscence Previous mesh infection
Clinical scenario	Emergency operation with bowel resection Intraperitoneal mesh removal Multiple hernia defects No primary closure possible without component separation

Table 19.2 Complex hernia severity defined by Slater et al.

Minor	Moderate	Major
One wound healing impairing factor[a]	Two or more wound healing impairing factors[a]	Two or more wound healing impairing factors[a] and one or more "moderate" class criteria
	Hernia >10 cm width or no primary closure possible without component separation	Surgical wound class III (Contaminated) or IV (dirty)
	Loss of domain >20%	Open (burst) abdomen
	Parastomal, lumbar, lateral, and subcostal hernias	Disease related: necrotizing fasciitis
	Full-thickness defects, loss of substance, distorted anatomy, or multiple hernia defects	Current mesh infection
	Skin grafts, wound ulcers, non-healing wound	Enterocutaneous fistula present
	Disease related: omphalocele	
	Increased intra-abdominal pressure: obesity or COPD	
	History of wound dehiscence or wound/mesh infection	
	Intraperitoneal mesh removal	
	Emergency operation with bowel resection	

[a]See Table 19.1: Obesity, diabetes, steroid use, smoking, old age, poor nutritional state (albumin <30 g/dL)

depends on several factors: the size of the abdominal wall defect (and the advancement needed for this defect), the presence of a stoma of any kind, the condition of the patient, and the preference of the surgeon.

Different techniques of advancement will be described below.

19.2.1 Rives–Stoppa Technique

Of all advancement techniques, the Rives–Stoppa is the least invasive technique. Described by Schumpelick et al., it is currently still being used for medium-sized defects where only a small advancement is needed [17–19].

In the Rives–Stoppa technique, a midline laparotomy is performed. If needed, the hernia sac can be opened and adhesiolysis can be performed. After this, the rectus sheet is opened. Then, the plane between the posterior rectus fascia and the rectus muscle is dissected to create space for a mesh. After dissection, the posterior fascia and peritoneum are closed using a running and slowly absorbable suture. Then, a mesh is placed in a retromuscular position and fixated if needed. Finally, the anterior fascia is closed using a running and slowly absorbable suture, and the subcutaneous tissue and skin are closed.

Ten studies show a recurrence rate of 3.6% after follow-up of 1–8 years [9, 20–24]. Postoperative wound complications were reported in 11% of all patients, seroma in 9%, and hematoma in 7%.

19.2.2 Anterior Component Separation Technique (Modified Ramirez Technique)

The anterior component separation technique was first mentioned by Albanese in 1951 [25]. In 1990, Ramirez et al. [2] described their component separation technique in a search for larger abdominal wall advancement. In the original anterior component separation technique, after dissection of the skin and subcutaneous fat, a relaxing incision is made in the aponeurosis of the external oblique muscle, separating the external and internal oblique muscle. Next, the rectus fascia can be incised to achieve greater advancement. Currently, this order is often changed, starting with dissection of the rectus fascia (release of rectus muscles from the posterior rectus fascia) and finishing with the separation of the oblique muscles. Originally, the operation was performed without mesh placement, but nowadays mesh augmentation is included in this technique.

The potential advancement of the bilateral anterior component separation technique has been studied, and results show a total advancement of approximately 5 cm at the level of the xiphoid process, 9–10 cm at the level of the umbilicus, and 3–8 cm at the level of the anterior superior iliac spine [2, 26].

Seven studies show that the anterior component separation technique is an effective treatment for large hernias with recurrence rates of 16% after mean

follow-up of 12–52 months [3, 5, 27–31]. Despite these results, postoperative complications occurred in 50% of all patients. Most frequent were infections or necroses of the wound, occurring in 20%, seroma in 9%, hematoma in 8%, and pulmonary complications in 7% of all patients. To prevent skin necrosis, it is paramount to avoid injury to the main subcutaneous perforator vessels at the umbilical level bilaterally.

19.2.3 Posterior Component Separation Technique (Transverse Abdominis Release or TAR)

The posterior component separation technique was developed as an alternative for the anterior component separation technique that leads to relatively high complication rates. The basic principle is the same: gaining advancement while retaining abdominal wall muscle coverage along the whole abdominal wall.

At first, like with the Rives–Stoppa technique, the rectus sheet is opened, and the posterior rectus fascia and rectus muscle are separated. At the lateral margin of the rectus muscle, the aponeurosis of the transverse abdominis muscle is incised. After this incision, the internal oblique muscle and the transverse abdominis muscle are separated, releasing the latter and creating advancement. The innervation of the rectus muscles should be respected as the intercostal nerves are running closely to the level of transection of the aponeurosis of the transverse abdominis muscle.

No studies have been performed investigating the potential advancement when performing the posterior component separation technique, but it is said to achieve advancement comparable to the anterior component separation technique.

19.2.4 Hernia Recurrence and Other Complications

Repair of large ventral hernias has relatively high recurrence rates up to 17% [9, 28, 32–34]. In patients with comorbidities such as obesity, diabetes mellitus, and chronic obstructive pulmonary disease, morbidity and recurrence rates can be up to 34% [28, 33–35].

Wound infection, seroma, hematoma, and skin necrosis are often observed after repair of large ventral hernias. Wound complication rates range from 13 to 48%. One of the causes of these high rates is the possible damage on the epigastric perforating arteries during dissection. This can compromise the blood supply to the skin and can interfere with wound healing [1, 28, 36]. Apart from these perforating arteries, the intercostal arteries can also be damaged, leading to comparable complications [5, 36].

Postoperative pulmonary complications are frequently reported, especially when there is loss of domain. This can require reoperation or prolonged ventilatory support [3, 37].

19.3 When Advancement is Not Sufficient: Other Options

19.3.1 Bridging

In some cases, the abdominal wall defect is too large (>15 cm) to facilitate tension-free closure. In these cases, mesh bridging is a possible treatment. Instead of direct fascial closure, a mesh is placed between the two fascial edges, bridging the present gap. The downside of this technique is the lack of mesh ingrowth in the absence of covering tissue and the risk of mesh infection. Despite these remarks, it can prove as an alternative for those defects too large to close with other techniques. In those patients, it is recommended not to excise the hernia sac but to use it as an additional barrier between the mesh and the subcutaneous layer and the skin.

In some cases where tension-free closure cannot be achieved, but bridging is not wanted, other options could be needed.

19.3.2 Pneumoperitoneum

When the defect size seems too large to close, preoperative progressive pneumoperitoneum can be of additional help. In 1947, Moreno was the first to describe the technique [38]. He used percutaneous punctures to inject intraperitoneal air. Since then, the technique has been modified, using catheters or subcutaneous ports to prevent the daily use of percutaneous punctures. The basic principle of the technique is simple. By gradually increasing intra-abdominal pressure, the abdominal wall muscles can stretch and adapt. In this way, the abdominal wall is prepared to an eventual surgery with tension-free closure.

There is no consensus on what gas to use or on the number of treatment days [39–43]. Most complications are directly related to the catheter or port placement (abdominal wall hematoma, catheter dislodgement, and peritonitis). Other rare complications can be subcutaneous emphysema or venous stasis in cases of high intra-abdominal pressure even with thrombosis of the caval vein.

A recent review identified 15 studies comprising 269 patients undergoing preoperative progressive pneumoperitoneum [44]. Fascial closure was achieved in 84% of all patients. After 1–48 months, recurrence was seen in 7.2% of patients and complications in 12%. Most common complications were subcutaneous emphysema and wound infection.

19.3.3 Botulin Toxin Injections

Used for other purposes, botulin toxin A injections cause local muscle relaxation. When injected in the abdominal wall preoperatively, it effects in the same way. The muscle relaxation creates additional advancement, allowing primary closure of the abdominal wall with one of the surgical techniques described before. It has been used for dystonias and spastic disorders of the abdominal wall

muscles, for pain management after laparoscopic hernia repair, but also for closure of large defects [45–51].

In 2005, Smoot et al. described the anatomic locations of six injections in the bellies of the three lateral muscles on both lateral sides, creating significant lateral muscle retraction [46].

A recent prospective study of 32 patients [52] showed promising results of this additional therapy with a mean advancement of 8 cm. This allowed all hernias to be closed without recurrence at a median follow-up of 19 months.

Take-Home Messages

- When tension-free abdominal wall closure after open abdomen is not possible, advancement is necessary.
- Depending on the needed advancement, procedure of choice can be Rives–Stoppa, anterior component separation (modified Ramirez), or posterior component separation (TAR) techniques.
- When advancement alone is not sufficient, bridging, pneumoperitoneum, and botulin toxin injections can be added to the treatment.
- Operation for large ventral hernias after open abdomen is a highly specialized surgery.

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Biological Prosthesis for Abdominal Wall Reconstruction

20

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Highlights

- No definitive evidences exist on the biological prosthesis in abdominal wall reconstruction after open abdomen; dedicated studies are needed.
- Biological prosthesis seems to be a valid option for abdominal wall repair minimizing mesh-related complications, especially in contaminated surgical fields.
- In managing great abdominal wall defects, the positioning of a biological prosthesis as a bridge to close the abdomen seems to be the best and most obvious solution to solve the acute problem.
- Biological prosthesis seems to be associated with a high rate of hernia recurrence in long-term follow-up.

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20.1 Introduction

Early definitive closure is an important goal in open abdomen treatment; there are some evidences showing better outcomes associated with early closure [1]. Primary fascial closure is the ideal solution to restore the abdominal closure, but sometimes the open treatment, particularly if prolonged, results in fascial retraction consequently in large abdominal wall defects that require complex abdominal wall reconstruction. Moreover the situation is often complicated by a contaminated surgical field with high risk of infections and wound complications, such as wound infections, seromas, fistula formation, recurrence of the defect, and mortality [2, 3]. Primary fascial closure failure in open abdomen has been reported ranging from 22 to 39% in a meta-analysis [4].

For these complex and troublesome situations, several techniques have been proposed and discussed comprehending the component separation techniques, flaps transpositions, and mesh repair. The abdominal wall defect that could result from an open abdomen treatment could be treated and managed with the aforementioned different techniques and with different timings. Depending on the center and surgeon's preference and expertise, combined with patient's conditions, open abdomen could result even in a planned ventral hernia, allowing the wound to granulate with or without skin grafting for the immediate closure and with a later planned abdominal wall reconstruction.

The aim of the present chapter is to give an overview of the use of biologic mesh in abdominal wall's reconstruction after open abdomen treatment, both in immediate "acute mesh repair" or in delayed planned ventral hernia repair.

20.2 The Rationale

Open abdomen, above all if prolonged, may result in fascial retraction and abdominal wall defects. The fascial defect could be closed with a mesh as a bridge. The main used nonabsorbable synthetic materials (i.e., polypropylene mesh) reinforce the fascial repair by a combination of mechanical tension and intense inflammatory reaction, resulting in the entrapment of the mesh into scar tissue. However synthetic meshes are generally not recommended on such situations [5]. The persistent inflammatory response may induce local side effects such as adhesions, erosions, and fistula formation, particularly when mesh is directly in contact with viscera [6–8]. Moreover in the presence of bacterial contamination, their use could result in a larger number of complications such as mesh infection and the need for mesh removal with related morbidity and mortality [9]. Among difficulties in the management of the open abdomen, there is the bacterial contamination. A recent paper reported that almost 80% of the patients with open treatment had a positive bacterial and fungal culture from the abdominal cavity [10]. All bacteria, whether in an acute or chronic wound or in a contaminated versus a colonized wound, produce virulence factors (e.g., exotoxins, endotoxins), with deleterious effects to wound healing [11, 12].

Because of the limitations of nonabsorbable synthetic meshes, surgeons have started to explore the use of biological materials in abdominal wall reconstruction. Biological prosthesis has been designed to perform as permanent surgical prosthesis in abdominal wall repair, minimizing mesh-related complications. There are several types of biological materials, derived from allogenic or xenogenic tissue, such as bovine pericardium, human cadaveric dermis, porcine small intestine submucosa, porcine dermal collagen, and bovine dermal collagen. For each material, several treatments are processed in order to remove hair, cells, cell components, and antigens present in the tissue, leaving only a highly organized collagen scaffold with the surrounding extracellular ground tissue [13]. After the implantation, biological responses lead to implant degradation and resorption with tissue remodeling in which the implanted material is repopulated by local fibroblasts and a new vasculature that together support the generation of a new, metabolically active, strong tissue [14].

The rationale of their usage in open abdomen is that the implantation of a biological material triggers a cascade of events leading to a new healthy tissue deposition and prosthesis remodeling. One of the most important aspects is that the collagen scaffold, repopulated with new tissue and blood vessels, allows blood, growth and pro-/anti-inflammatory factors, and drugs to reach the surgical field during the first phases of healing process. The presence of vital tissue allows therefore preventing mesh infection and abscess formations, maintaining mechanical characteristics of a synthetic mesh with a sufficient mechanical strength to withstand the physiological and anatomic stresses of the human abdominal wall.

20.3 Prosthesis Choice and Position

Natural cross-links exist in native collagen, and their function is to stabilize the structure of the collagenic proteins, giving mechanical strength and protection from collagenase. Biomaterials, during the preparation phase, could be treated with the use of chemical cross-linking agents modulating the characteristics of the prosthesis and giving additional strength to the collagen scaffold. The presence of cross-links between the collagen chains seems to further reduce the bacterial and host collagenase enzymes activity, slowing the degradation process of the prostheses. Therefore cross-linked prosthesis is partially remodeling, whereas non-cross-linked prosthesis is completely remodeling. Despite each prosthesis which permits and encourages host tissue ingrowth, the differences in remodeling times should be kept in mind when considering the choice of the best prosthesis. Even though it has been demonstrated in animal models that the tensile strength is different between cross-linked and non-cross-linked meshes during the first months after the implant, both type of materials show similar values after 12 months with no changes over time in the strength of the repair sites [9, 15–17]. The choice of biological materials in contaminated fields is recommended by the Ventral Hernia Working Group and the WSES guidelines on emergency abdominal wall repair [5, 18]. In literature a decisional model proposed by “The Italian Biological Prosthesis Work Group

Table 20.1 IBPWG score = infection score × tissue loss score

Infection:	Score	Tissue loss	Score
Potentially contaminated	1	No tissue loss	1
Contaminated	2	0–5 cm defect	2
Infected	3	>5 cm defect	3

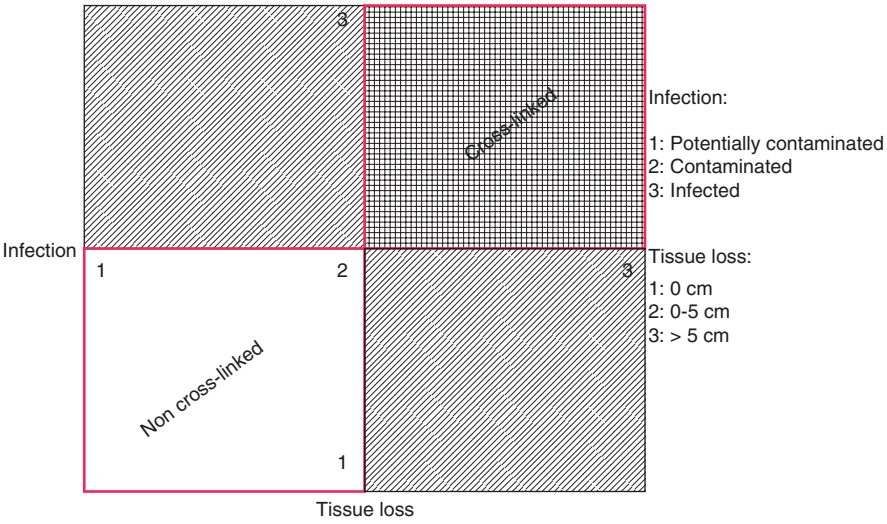

Fig. 20.1 Decisional diagram of IBPWG based on infection and tissue loss. Reproduced with permission from [20]

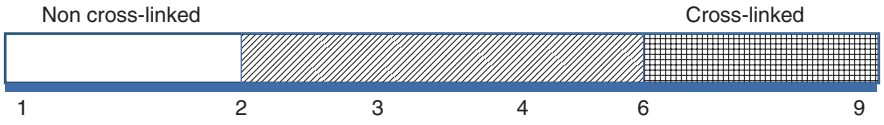

Fig. 20.2 Decisional model based on IBPWG score (on the x-axis) (Reproduced with permission from [20])

(IBPWG)" is available also in order to standardize and to facilitate the choice between the different types of BP [19]. The proposed model combines the tissue loss' dimension and the contamination of the surgical field resulting in a score that indicates the necessity to use either a cross-linked or a non-cross-linked BP (Table 20.1, Figs. 20.1 and 20.2).

No good quality data exists on prosthesis position in closure of the abdominal wall. Moreover available evidences evaluate each prosthesis' position combined with various and heterogeneous prosthesis (synthetic and biological) in heterogeneous patients. A recent retrospective study on porcine cross-linked dermal prosthesis with a long follow-up showed better results with sublay position compared with onlay (2.4 versus 18.9% recurrence, $p < 0.0001$), but data were not specific on an

open abdomen [21]. Two meta-analysis confirmed that the sublay position results in a lower infection rate and recurrence rate compared to onlay, inlay, and underlay [20, 22], but it should be stressed that included data were not specific on biological prosthesis and open abdomen and the heterogeneity among patients and indications was very high, giving a poor level of evidence.

Discordant data have been published about the use of BP to bridge wide defect of the abdominal wall. Few studies, nonrandomized, and with small number of cases and heterogeneous patients, have been published reporting recurrence rate ranging between 0 and 100% [15, 16, 23–25].

In closing the open abdomen, the mesh is often required not only to reinforce the abdominal wall and to prevent incisional hernia but also as a bridge to close the absent/retracted fascia. No studies dedicated specially on this issue are available in literature right now, and data could be only extrapolated from larger studies. A large study on the use of acellular porcine dermal collagen in hernia with high risk for infections reported an overall recurrence rate of 66% [26]. When the meshes were positioned as a bridge, the reported recurrence rate was >80%. A study by Booth and colleague compared primary fascial closure with mesh reinforcement with the use of the mesh as a bridge and demonstrated a higher recurrence rate (56% versus 8%, $p < 0.001$) [25]. A randomized controlled trial compared the traditional bridged mesh repair (with both synthetic and biologic meshes) with the component separation technique with biologic mesh reinforcement [27]: the trial demonstrated that recurrences were lower with the component separation plus mesh reinforcement technique (13.2 versus 37.5%, $p = 0.02$). Component separation plus reinforcement was also associated with a lower infection rate (0 versus 23% in the bridged group, $p = 0.002$), but it should be noticed that in the traditional bridged mesh repair group, synthetic meshes were also included and all the infective events occurred within this subgroup.

20.4 Results

As aforementioned lots of studies about the use of biological prosthesis in abdominal wall reconstruction exist in literature with different and contrasting results [24, 28–44]. A great lack of evidence exists on the long-term results. A retrospective study by Rosen et al. [45] included 128 patients with ventral hernia and a contaminated field treated with a biological prosthesis. They showed a rate of complicated wound of 47%, and they demonstrated a recurrence rate of 31% after a mean follow-up of 21 months. Great limitations of the study were the utilization of different kinds of prostheses in different positions (inlay, onlay, etc.) and with the combination of the component separation technique.

A meta-analysis of 2012 reported data with a comparison among different types of meshes [46]: the recurrence rate was similar for cross-linked and non-cross-linked porcine dermis (10% versus 8%) but was significantly higher for allogenic human dermis (recurrence rate 20%). Wound complications were similar among the three different types of meshes.

A systematic review by Bellows et al. [47] reported data of 1212 patients. With a mean follow-up of 13 months, the reported weighted recurrence rate was 15.2%. Among the complications, postoperative infection and seroma were the most common with a weighted incidence of 16.9% and 12%, respectively. The removal of the prosthesis was reported in 2% of the cases due to poor mesh incorporation in the majority of cases. Cross-linked porcine meshes result in similar recurrence rate (11% versus 10%) and lower infection rate (9% versus 18%) compared to non-cross-linked meshes.

A meta-analysis by Lee et al. [36] investigated biologic meshes compared with synthetic meshes in abdominal wall reinforcement in contaminated fields. Short-term results as wound infection, prosthetic explantation, and enterocutaneous fistula were comparable in the two groups; biologic meshes were associated with a higher incidence of hernia at follow-up.

A recent meta-analysis by Sharrock and colleagues investigated the management and closure of open abdomen in trauma patients [48]. Among the included studies, the point estimate recurrence rate of ventral hernia after 1 year of biologic mesh positioning was 51%. However, the authors highlighted the small number of included studies and their poor quality, suggesting great caution in interpreting this result.

A systematic review and meta-analysis by Atema et al. [49] investigated the utilization of biological material in abdominal wall reconstruction; the poor quantity and quality of available data strongly limits the results. Biological materials in infected fields had a recurrent hernia rate of 30% compared with 7% of synthetic materials, but data were derived from a single study and do not justify the use of synthetic materials, especially as a bridge position after open abdomen.

Available evidences are really weak: all the cited meta-analysis included especially poor-quality retrospective case series. There is also a great heterogeneity among indications for mesh implantation, mesh position, and type of mesh. This further weakens the quality of the evidences. Actually there is no randomized trial comparing different types of meshes or the indication to mesh positioning. Moreover no good quality comparative studies dedicated on closure of open abdomen are available. Several randomized controlled trials are ongoing to assess the safety and long-term results of biological prosthesis in abdominal wall reconstruction even if none of them is dedicated exclusively to open abdomen [50–58].

Conclusion

No definitive evidence-based conclusions could be obtained right now from the literature, and no clear indications in specific situations could be defined for the use of biological prosthesis in abdominal wall repair. Biological prostheses have been designed to perform as a valid option for abdominal wall repair minimizing mesh-related complications, especially in contaminated surgical fields. In managing great abdominal wall defects, especially after open abdomen, biological prosthesis are a fundamental part of the armamentarium of our surgical practice and remains “the only option” in some troublesome situations despite the lack of robust evidences. The need for consensus on the role of biologic mesh in abdominal wall reconstruction is evident. Randomized trials are difficult to conduct,

especially in open abdomen, and so prospective studies or large registries are needed with uniform definitions and inclusion. At the moment, the positioning of a biological prosthesis as a bridge to close the abdomen seems to be the best and most obvious solution to solve the acute problem, keeping in mind the possibility to hernia recurrence in long-term follow-up.

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Rehabilitative Process, Functional Impairment, and Quality of Life

21

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Key Points
- Early mobilization
- Respiratory exercise
- Strength training program
- Functional activity
- Eventually orthosis

21.1 Introduction

In literature, only two studies exist about physiotherapy rehabilitation in patients undergone to open abdomen procedures in emergency and trauma surgery [1, 2].

The first is an article published in 2002 by Mackay et al. [1] investigating clinical outcomes and physiotherapy staffing resources in open abdominal surgery patients in two hospitals. They designed an evaluation questionnaire, called Abdominal Surgery Physiotherapy Outcomes Data Sheet (APODS), which demonstrated good content validity and high intrarater and interrater reliability. Clinical outcomes achieved and the staffing resources required were compared for 30 high-risk open abdominal surgery patients at two metropolitan hospitals. Both hospitals were similar in preoperative morbidity, demographics, and operative characteristics. The results of this study indicate that open abdominal surgery patients have the potential to consume very high levels of physiotherapy resources in terms of staff time and

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days of treatment, with a large proportion developing clinically significant postoperative complications. The APODS demonstrated good content validity, and high kappa values in virtually all data categories, indicating good reliability, particularly in the application of indicators for sitting out of bed, day first walked, and scoring of postoperative pulmonary complications (PPCs). Development of clinically relevant outcome measures such as those described can provide objective data for guiding staffing, future service planning, and setting research priorities.

Lately, the same group [2] conducted a study aiming to determine if the addition of deep breathing exercises and secretion clearing techniques to a standardized physiotherapist-directed program of early mobilization improved clinical outcomes in patients undergoing open abdominal surgery.

This study suggested that, in this clinical setting, the addition of deep breathing and coughing exercises to a physiotherapist-directed program of early mobilization does not significantly reduce the incidence of clinically significant postoperative pulmonary complications in high-risk open abdominal surgery subjects.

This study found that, in this clinical setting, the addition of coached lateral basal expansion and secretion clearance techniques to a targeted program of physiotherapist-directed early mobilization conferred no additional benefit in reducing the incidence of postoperative pulmonary complications after open abdominal surgery in high-risk subjects.

This study was significant in that, unlike previous randomized trials of physiotherapy after open abdominal surgery, it documented the actual type and dosage of physiotherapy interventions and used a sample of subjects from the population of open abdominal surgery patients who are most likely to benefit from physiotherapy, that is, those identified as high risk of developing postoperative pulmonary complications.

These results were obtained in a clinical setting where patient-controlled epidural analgesia was used extensively and the standardized early mobilization program was implemented and monitored by the physiotherapists.

Due to the lack of literature in the rehabilitation after open abdomen procedures in emergency surgery, we can investigate other kinds of abdominal surgery to derive a rationale to apply in rehabilitation programs after and during open abdomen procedures.

Many authors talk about surgery interventions that allow patients to conduct some preoperative physical activities in order to undergo surgery in the best physical condition; as far as open abdomen surgery is concerned, this kind of prevention is not possible because it is often an emergency condition.

In the immediate postoperative period, the typical breathing pattern of the patient is small tidal volume, increased respiratory rate, and a weak cough leading to retained secretions, which is thought to be the main contributor to PPCs [2]. It is important to encourage the correct breathing pattern for lung volume expansion, particularly in patients with COPD, as this should assist with secretion clearance. Moreover, after surgery, patients experience tiredness, lethargy, changed emotional state, and a desire to sleep and rest, more commonly known as “postoperative fatigue.” [3]

The aim of physiotherapy, for patients after open abdominal surgery, consists of a variety of interventions intended to improve cardiopulmonary and/or physical function, reverse physiological and/or functional changes, reduce the incidence of PPCs and intensive care unit and hospital stays, improve quality of life, and regain preoperative independence in all the activities of daily living.

These interventions may include lung expansion exercises, secretion clearance techniques, limb exercises, progressive mobilization programs, and other techniques [2, 4].

21.2 Common Respiratory Complications of Abdominal Surgery

PPCs are the most significant cause of morbidity following open abdominal surgery [1], despite advances in perioperative care [2], and are responsible for the increased mortality as well as length of hospital stay and health-related cost of care [5, 6]. PPCs have been defined as "an identifiable disease or dysfunction that is clinically relevant and adversely affects the clinical course" [7].

Atelectasis, pneumonia, acute respiratory failure, tracheobronchitis, wheezing, and prolonged mechanical ventilation are most commonly observed PPCs [8]. It is known that the decrease in lung volumes and capacities, abnormal respiratory pattern, abnormal gas exchange, and pulmonary defenses in patients undergoing open abdominal surgery start with anesthetic induction and perpetuate in the postoperative period, contributing to the occurrence of these PPCs. The respiratory muscle dysfunction has also been attributed to the development of PPCs [9]. Multiple factors may be involved in diaphragmatic dysfunction, such as irritation and inflammation caused by trauma from manipulation close to the diaphragm, reflex inhibition of afferent abdominal receptors, and postoperative pain. [4].

The incidence of postoperative pulmonary complications has been shown to be lower in open abdominal surgery patients who receive physiotherapy compared to those who receive none [2, 10, 11].

In some cases, such as low blood pressure, severe premorbid debility, patient refusal, pain, atrial fibrillation or other cardiac arrhythmia, nausea and vomiting, motor block from epidural, nurse concern, low hemoglobin, or others [12], physiotherapy should be delayed in order to permit patient's clinical stabilization.

Criteria for postoperative pulmonary complications [12]:

1. Chest radiograph report of collapse/consolidation
2. Raised maximal oral temperature >38°C on more than one consecutive postoperative day
3. Pulse oximetry oxygen saturation (SpO2) <90% on more than one consecutive postoperative day
4. Production of yellow or green sputum different to preoperative assessment
5. Presence of infection on sputum culture report

6. An otherwise unexplained white cell count greater than 11 × 109/l or prescription of an antibiotic specific for respiratory infection
7. New abnormal breath sounds on auscultation different to preoperative assessment
8. Physician diagnosis of postoperative pulmonary complication

21.3 Measurement

Having the right evaluation instruments is fundamental in order to monitor both the treatment and the conditions of the patient.

Since clinical decisions about patient management incorporate a wide range of factors, practical approaches are required to assist clinicians in making the optimal management decisions [7].

Outcome measures most frequently used are temperature, expectoration, subjective experiences and time to chest tube removal, postoperative complications, alveolar–arteriolar oxygen difference and cough, dyspnea, mobilization, pain, use of bronchodilators, antibiotics, oxygen, pulse rate, and pulmonary auscultation [13].

21.4 Risk Factors

Among all possible risk factors for developing PPCs, there are age greater than 59 years, female gender, body mass index (BMI) greater than or equal to 27 kg = m2, lung disease, history of smoking, hypertension, diabetes, dyslipidemia, heart disease, cancer, type of surgery, surgical technique, time of surgery, and surgical risk (American Society of Anesthesiologists scale) [4].

Decreased cognition also has been cited as a risk factor for pulmonary complications due to the decreased ability to protect their airway [1, 14].

The primary modifiable risk factors are shallow breathing, incision pain, and immobility. Breathing exercises and early mobilization are cornerstones of postoperative management. Early mobility following surgery is deemed crucial, since postoperative immobilization is widely held to contribute to cardiovascular instability, thromboembolic complications, and catabolism, in addition to pulmonary morbidity [3].

21.5 Treatment

In literature all the studies agree on the application of physiotherapy in the post-intervention period. Patients are included in standardized programs of physical therapy treatment which structured the model of patient care, focusing on the use of additional therapeutic resources [4, 15, 16], early sitting position, and ambulation (onset <48 h after surgery) [4, 17].

The reported studies show the comparative effectiveness of different treatment modalities including early mobilization (EM), ambulation, incentive spirometry, continuous positive airway pressure (CPAP), positive expiratory pressure (PEP), and conventional physiotherapy including deep breathing exercises (DB&C) [2, 7, 10, 18].

The DB&C exercises consisted of at least three coached lateral basal expansion maneuver (deep breaths) followed by a cough, huff, or forced expiratory maneuver in order to facilitate sputum clearance [2].

Physical therapy modalities are generally left to the discretion of the therapists, provided that they correspond with guidelines regarding abdominal strengthening and stabilization, abdominal myofascial release and scar mobilization/massage, core strengthening in neutral only (no crunches), balance training, hip mobilization, gluteus medius strengthening, lumbar strengthening, posture retraining, and upper back strengthening. Physical therapy is recommended—at least two sessions per week for 6 weeks—and encourages a return to independence with regard to mobility and activities of daily living [19].

Early mobilization (EM) has been shown to improve forced vital capacity. Sitting out of bed has been shown to be associated with an increase in postoperative functional residual capacity [20], and it has been suggested that a program of active enforced progressive mobilization can improve pulmonary function [21]. The results of one study comparing EM with EM + DB&C demonstrated that, despite having fewer PPCs, the group who received delayed mobilization (from third postoperative day) and deep breathing exercises needed more physiotherapy input and took longer to be discharged from physiotherapy compared with the early mobilization group [1]. Also, delayed mobilization can increase the risk of PPCs [22]. Patients with severe COPD may have difficulties with walking [3].

The reported clinical benefits of early mobilization of mechanically ventilated patients are a decreased duration of mechanical ventilation, reductions in the incidences of ventilator-associated pneumonia (VAP) and intensive care unit (ICU) delirium, shorter ICU and hospital stays, and improved functional status upon hospital discharge [23].

In the immediate first period after surgery, it is important to encourage people to move in the right way in order to prevent fear of movement, pain, and lung volume reduction. Changes in body position and physiotherapy aimed to airways clearance are the most necessary interventions. In a second postoperative phase, when the patient has finally reached stable clinical conditions, it is important to focus on physical activity in order to recover a complete independence in all daily living activities [1, 2, 7, 20, 23–25].

21.6 Phase 1

Mobilization in postoperative patients may be defined as low-intensity exercise that aims to elicit cardiopulmonary responses which enhance oxygen transport and assist in the reduction of postoperative pulmonary complications [25, 26].

A study carried out on patients after upper abdominal surgery found that FVC increases progressively between supine at 0° and upright positions. This is the first study evaluating respiratory mechanics through VC in this type of surgical patients, which is somewhat relevant, as the upper surgeries predispose to complications and positioning may minimize some ventilatory changes. In the present study, the sitting position showed an increase of 20.2% in FVC compared to

Table 21.1 Evaluation of the forced vital capacity (FVC) in liters (L) in different body position, with 95% confidence interval (95%CI) modified from Martinez et al. [25]

Body position	Mean	Standard deviation	Confidence interval (95% CI)
Supine (0°)	1.68	0.47	1.51–1.85
Supine (45°)	1.86	0.48	1.68–2.04
Sitting	2.02	0.48	1.84–2.21
Upright	2.18	0.52	1.99–2.37

supine position. This finding corroborates other studies that showed increased FVC in this position ranging from 4.6 to 20% in patients undergoing abdominal procedure (Table 21.1) [25].

Nielsen et al. conducted a review in which the effect of posture on postoperative pulmonary function was studied [20]. They found out that the sitting and semirecumbent positions compared with the supine position in six of 12 studies improved postoperative pulmonary function. However, a change from the supine to lateral position in patients in the intensive care unit showed a limited effect on pulmonary function. The duration of the mobilization has not been considered, including repeated measurements over days. The physiologic effects of postoperative immobilization have never been thoroughly examined, but are widely supposed to add to thromboembolic and pulmonary complications as well as catabolism and cardiovascular instability [27]. Although most agree that postoperative immobilization should be avoided, standard care often includes physiological immobilization for several days [28]. The turning of patients from the supine to lateral position is a routine practice in the postoperative intensive care unit. Despite the lack of evidence of a substantial beneficial effect on pulmonary function revealed in this review, it is still recommended in respect to decubitus prophylaxis and prevention of atelectasis and deep vein thrombosis [27].

The criteria used to assess patient suitability for mobilization are:

- Patient awake
- Stable blood pressure
- Stable heart rate
- No dyspnea at rest

Pain score <8 on visual analogue scale [2, 7, 24].

21.6.1 Mobilization

The study conducted by Zafiropoulos et al. [26] reported a progression from body position changing to walking in a sort of circuit that they called the Ayres circuit: supine, sitting over the side of the bed (by rolling onto the side and pushing up into sitting), standing, walking on the spot for 1 min as able (the number of steps taken was recorded), initially after sitting out of bed in a chair [26].

Mobilization, as defined in this study, resulted in significant increases in VT, *f*R, and VE when patients moved from supine to standing. No further statistically significant increases were observed in these parameters with walking on the spot for 1 min [26].

The progression of activity to walking on the spot for 1 min, in this group of patients, favored an upper chest pattern of breathing with significant increases in VT/TI. Hemodynamic changes were mainly observed as increases in blood pressure when the patients initially sat over the edge of the bed. No statistically significant changes were observed in arterial blood gas parameters after mobilization [26].

To sum up, it is essential to position patients in a stable, supported upright sitting position and to teach them to huff/cough with wound support as soon as possible postoperatively. Further a prophylactic physiotherapy intervention following abdominal surgery was essential. The choice of intervention could either be mobilization or breathing exercises, but does not need to include both options.

Furthermore, it is essential that when breathing exercises are prescribed, they should be performed frequently.

In literature, there are few studies that analyze and describe respiratory physiotherapy after open abdominal surgery. Some of them talk about a program of early mobilization and leg exercises as the one commonly described for the non-*coached lateral basal expansion exercises and sputum clearance techniques*, and they introduce the concept of deep breathing and coughing (DB&C) exercises. The DB&C exercises consist of at least three coached lateral basal expansion maneuvers (deep breaths from functional residual capacity to total lung capacity, with a 3-second inspiratory hold followed by relaxed expiration) followed by a cough, huff, or forced expiratory maneuver. Instruction and supervision from the physiotherapist focus on bilateral basal expansion, avoiding upper chest and shoulder elevation and maximizing expansion of the lower chest diameters during inspiration, with a 3 s end-inspiratory hold, followed by relaxed expiration. This is done with the subject in sitting with the physiotherapist providing bilateral proprioceptive feedback with the hands on the lower ribs. This cycle is to be repeated at least twice during each treatment. Patients are also encouraged to practice these DB&C exercises every waking hour by themselves.

All the above interventions could be administered by the ward physiotherapists three times daily on postoperative days and then daily until the patient is independently mobile and had a clear chest assessment for three consecutive days. A clear chest was defined as no auscultation or chest radiograph changes, normal temperature, and no sputum [24].

The importance of coughing is supported by strong evidence from the systematic reviews [3, 24] in patient undergoing upper abdominal surgery. It would be essential also to include management strategies to ensure effective secretion removal. These strategies are based on the collective clinical experience. It includes positioning the patient out of bed and using interventions such as deep breathing, positive expiratory pressure (PEP), high-pressure PEP, and CPAP in combination with forced expiration technique (or adjusted autogenic drainage) [7].

Other studies [13, 24] present simple systems for positive expiratory pressure (PEP) breathing, for example, systems where a mask or a mouthpiece is connected to a resistance nipple to provide positive pressure during expiration and the blow-bottle device in which the resistance consists of a water seal. In clinical practice, the resistance is often regulated to achieve 5–20 cmH_2O during slightly active expiration. The pressure achieved is dependent on the performance of the maneuver, the adjustable expiratory resistance, and the patients' active expiratory flow. The rationale for the PEP technique postoperatively is to increase pulmonary volumes, decrease atelectasis, and promote secretion removal [7, 8]. The physiological effects of PEP in spontaneously breathing postoperative patients are unknown, but an increased functional residual capacity (FRC) is considered essential [13].

In the study of Orman et al. [13], conventional chest physiotherapy consisting of breathing exercises and postural drainage is compared with the blow-bottle technique, performed for 10 min every fourth hour.

The authors show that the addition of 20 breaths performed with a blow-bottle system (110 cmH_2O) every second hour in addition to standard pre- and postoperative physiotherapy did not show any significant improvements [13].

Further they compared the performance of 30 breaths every waking hour, with CPAP and with a control group receiving basic physiotherapy with incentive spirometry. The alveolar–arteriolar oxygen difference was significantly lower, and PaO_2 and forced vital capacity were significantly higher compared to the control group on the second postoperative day. Furthermore, atelectatic consolidation was significantly lower in the PEP group on the third postoperative day [13].

The least likely choice is incentive spirometry (IS) [7, 29] followed by intermittent positive pressure breathing (IPPB). Both these techniques are dependent on specialized equipment and therefore costly to the patient. Two systematic reviews reported no added benefit to deep breathing exercises [7, 30]. In addition, IPPB would be the last choice because abdominal distention has been reported as potentially harmful and the technique is therapist dependent [7, 29, 30].

Respiratory physiotherapy techniques used for mechanically ventilated patients are treated in a very similar way and are divided into three activities: mobilization, chest physiotherapy, and muscle retraining. The procedures of mobilization include posture improvement, passive and active limb exercises, and continuous rotational therapy [23, 31].

Recent studies have demonstrated the efficacy and safety of advanced mobilization programs, which include additional elements such as prone positioning [23, 32, 33] or 45° rotation in the prone position [34], early exercise using a bedside bicycle ergometer [35], and walking exercises [36]. These additional approaches increase the range of intervention criteria and programs of early mobilization [23].

21.7 Phase 2

When the patient has finally reached stable clinical conditions, and the prosthesis is closed, it is important to focus on physical activity in order to recover a complete independence in all daily living activities.

In this phase, it is important to motivate the patient to reach some goals during each treatment session:

- Sit out of bed.
- Walk 5 m with assistance.
- Walk 15 m with assistance.
- Walk 30 m with assistance.
- Walk 30 m without assistance.

In the study conducted by Mackay et al. [2], subjects were encouraged to achieve one or more goals during each treatment and were encouraged to walk at a speed where they were taking deeper breaths than at rest, at an intensity of at least 6/10 according to the Borg ten-point scale of perceived exertion [37, 38]. This was intended to challenge the respiratory system sufficiently to produce an increase in minute ventilation. Subjects were permitted to also attempt the goals with the nursing staff and with visitors. Subjects were encouraged to perform active ankle planter flexion and dorsiflexion exercises, at least 20 times every waking hour, while in bed [2].

In the study of Arbane [22], subjects belonging to the exercise group received both standard care and a once-daily cycle (aiming for 30 min/session) and strength training. Strength training was based on the ten maximum REP principle (maximum amount of weight one can lift in ten repetitions) using appropriate ankle weights. The cycle component of the exercise program consisted of a 2-minute familiarization period of unloaded pedaling at 50–60 revolutions/minute (rpm), with the intensity increased steadily during the third minute to achieve a maximum of 60–90% of heart rate (HR) reserve [target HR range = (HRmax − HRrest) × percent intensity] + HRrest (taking account of the following correction factor: 12/25% reduction in preoperative maximum load for lobectomy/pneumonectomy) [22].

In the period following surgery and after being dismissed from hospital, activity restrictions are eased, and patients may lift items weighing 10–15 pounds. The walking regimen is also increased to 30 min daily or more and to the extent that a patient should be able to have a conversation without feeling short of breath. At this stage, isometric abdominal exercises are initiated, which assist in rebuilding core abdominal wall muscles.

Only later patients are allowed to lift objects heavier than 15 pounds and encouraged to begin physical therapy. At this stage, they begin a graduated return to full activity. Every month thereafter, patients will be able to lift ten additional pounds, to a target between 50 and 70 pounds, depending on the patient's preoperative functional assessment [19].

21.8 Orthosis

A study conducted by Cheifetz et al. [3] tries to investigate the effect of abdominal support on functional outcomes in patients following major abdominal surgery (MAS).

The question was whether early mobilization and pain control of patients following MAS can be facilitated by the use of an elasticized abdominal binder that

surrounds the abdomen and supports the incision [3]. Starting on POD 1 (postoperative day 1), patients in the experimental group were fitted with a binder that was applied prior to the first morning transfer and worn at all times when out of bed, including during ambulation. The elasticized binder was applied over the abdominal surgical incision, with the upper border not higher than the lower margin of the rib cage, ensuring minimal restriction of lateral costal expansion and diaphragmatic excursion. In addition, for participants who had stomas, drains, or other lines or tubes inserted during surgery, holes were cut in the binder to ensure that no pressure was applied over these devices. Patient comfort determined the tension of the binder; however, for maximal benefits to be derived from binder support, it was applied firmly (binder circumference 10–20% smaller than the patient's postoperative abdominal circumference measured at the level of the umbilicus) [3].

Results from this study suggest that the use of an elasticized abdominal binder after major abdominal surgery can enhance the speed of postoperative recovery of walk performance. Furthermore, the study demonstrated that circumferential support of abdominal incisions results in less distress from common postoperative symptoms, with an important reduction in pain, which suggests a less stressful experience after surgery. Importantly, the safety of postoperative binder use was attested by the results of the pulmonary function tests, which demonstrated that the elasticized abdominal binders did not adversely affect lung function [3].

In view of the noted beneficial effects on enhancement of early mobilization, pain control, and emotional distress, and in the absence of adverse side effects, study results suggest that use of an elasticized abdominal binder warrants consideration by clinicians in the postoperative management of individuals undergoing major abdominal surgery [3].

Recently a study who tried to find a consensus on the physiotherapeutic management of patients following upper abdominal surgery proposed to "Perform a clinical evaluation of pain level" before starting mobilization. The authors agreed that prophylactic physiotherapy intervention following abdominal surgery was essential, but that the choice of intervention could either be mobilization or breathing exercises, but does not need to include both options [7].

But in a field with a lack of studies, the importance of clinical expertise in the clinical decision-making process has been reported [38]. However, ways in which this expertise can be defined and incorporated into evidence-based practice still need to be explored, specifically in gray areas of clinical practice [7].

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Results of Treatment with an Open Abdomen and Future Directions

22

Dieter G. Weber and Sana Nasim

22.1 Introduction

The open abdomen, or laparotomy, has become an accepted, standard concept in contemporary surgical practice [1–6]. However, its indication for use remains heterogeneous, and the subtleties around its practical management remain equally diverse. These varying indications, in combination with stark management differences, continue to pose challenges in modern practice and complicate direct comparison of outcomes.

The open abdomen was first described in non-traumatic pathologies [7–11]. By the 1980s, several case series emerged, suggesting benefit in patients with severe and complicated peritonitis [7–11]. However, these initially sporadic reports were initially largely disregarded, and primary definitive operative strategies prevailed as the dogma of good surgical management. In spite of this historic standard, during the 1980s, several trauma centers also observed marked improved survival with initial abbreviated, temporizing surgical strategies, coupled with intensive physiological resuscitation, and delayed definitive surgical repairs, for their most physiologically deranged patients [12, 13]. This emerging, damage control, surgical strategy provided the laparotomy procedure with its now classic application. Developed just before the epidemic rise in incidence of the abdominal compartment syndrome (ACS) associated with the excessive saline resuscitation techniques of the 1990s and early 2000s [14, 15], the damage control strategy, with incorporation of the laparotomy, provided the timely and ideal tool for surgical practice.

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However, as had already been hinted by the early case reports and series of the 1980s, an increasing body of more robust evidence has emerged during the past decade, supporting the application of the open abdomen in a diverse range of non-traumatic abdominal emergency surgeries [16]. These indications largely relate to pathologies resulting in (1) intra-abdominal hypertension (IAH) or a frank abdominal compartment syndrome (ACS), (2) the need for multiple re-exploration of the coelomic cavity, or (3) the loss of the abdominal wall for a diverse range of reasons. The open abdomen is not a concept unique to trauma surgery; however, it was perhaps trauma surgeons that championed its application into the other sub-specialities, and the mainstream, of general surgery.

In this chapter we review the outcomes of the open abdomen. We first discuss the potential benefits of the procedure depending on the pathologies being treated. Thereafter, we address the potential complications, which are also associated with the approach, and the strategies that may assist offsetting this potential harm. We conclude with a discussion surrounding potential future directions for both clinical application and research.

22.2 Benefits of the Open Abdomen

22.2.1 Trauma

A select group of patients undergoing a trauma laparotomy benefit from an open abdomen [6, 17–19]. However, no randomized controlled data exist to guide this selection or to guide who will benefit the most. The primary data supporting the beneficial outcome from these laparotomies is in the form of carefully described case series [12, 13]. In these cohorts, marked improved survival had been demonstrated through their incorporation into the overall treatment strategy; the benefits are largely implied and extrapolated from the reported benefit of the overall damage control treatment modality. Otherwise, the purported benefits are suggested as self-evident, i.e., situations of necessity, where no alternative exists or where harm exists from the alternative treatment strategy (e.g., a tensioned closure).

The majority of trauma patients treated with a laparotomy receive this intervention as part of a damage-control surgical strategy [3, 6, 17]. In this situation, the open abdomen avoids IAH or an ACS (associated with a major increase in otherwise avoidable mortality) and facilitates planned re-exploration and definitive surgery. However, it should be noted that a damage control strategy is not an absolute indication for an open abdomen and, at times, may still be inappropriate. As discussed later in this chapter, potential harm from an open abdomen must be balanced against the expected benefits; there are few data other than to suggest a tailored surgical approach, exercising the best judgment of the trauma surgeon. Occasionally, other indications, such as a traumatic abdominal wall injury, or gross contamination necessitating repeat debridement, may also exist in the trauma setting. In any case, the number of laparotomies is small; only around 10–15% of patients undergoing a trauma laparotomy require a damage control approach [6].

Patient selection is critical to the realization of the reported benefits of the laparotomy. This is important for two reasons: first, the correct patient is required to benefit, but second and perhaps more importantly, given the potential larger number of patients in this potential group, the incorrect patient will be exposed to significant harm by inappropriate application of this technique.

An empiric laparotomy is indicated in patients undergoing a damage control strategy from traumatic abdominal injury, particularly, when reoperation during the next 24–48 h is required, and in the presence of severe visceral edema. A laparotomy should still be considered in patients who do not warrant a therapeutic laparotomy but in whom an ACS develops and has failed to respond to nonoperative measures [20, 21].

A laparotomy has also been more controversially considered for patients with refractory intracranial hypertension associated with multiple compartment syndromes [22]. However, further research is required before making any definitive conclusions in this regard.

22.2.2 Intra-abdominal Sepsis

Patients with severe intra-abdominal sepsis may potentially benefit from an open abdomen in several respects: (1) it may form part of a damage control operative strategy; (2) it may facilitate the concept of a "relook," i.e., allow repeated abdominal exploration in patients with gross contamination; (3) it may avoid the formation of an ACS; and (4) it has been proposed that the rate of stoma formation may reduce (the second stage of the procedure may facilitate a safe time for anastomosis) [1, 3–5, 16].

As for traumatic indications, the role for the open abdomen in the setting of a damage control operative strategy is based largely on case series [6]. An increasing number of supportive experiences have recently been published, describing the cautious extension of clinical damage control experience from trauma surgery to nontraumatic abdominal emergencies [16]. In analogous fashion with trauma patients, a small subgroup of patients in septic shock are insufficiently hemodynamically stable and have reach a point of physiological exhaustion, necessitating abbreviated surgeries, and benefit from aggressive intensive care efforts, before protracted restorative surgeries are undertaken. However, in contrast with traumatic shock, we have proposed elsewhere [16] that an additional initial phase of resuscitation should be incorporated into the traditional damage control sequence in the case of abdominal sepsis. In order to materialize any potential benefits, similar attention to patient selection is required, as well as care exercised to avoid its overuse.

Repeated laparotomies for debridement and peritoneal washout may benefit patients with the most severe intra-abdominal infections by attenuating the inflammatory mediators present in the peritoneal fluid, where infected collections develop or where incomplete debridement has occurred [1–5]. A planned relook in 48 h is easily facilitated by the open abdomen. However, once mainstream, the concept of "relook" laparotomies has been studied with both randomized data and large case

series [1, 23, 24]. Given similar mortalities experienced in either treatment methods, mandatory "relook" policies have given way to "on-demand" strategies. Vigilant observation, and immediate reoperation in the setting of deterioration, is required for the "on-demand" approach. However, unfortunately the precise clinical triggers governing returns to theater are not well defined and continue to be the topic of research [24].

Significant improvements in the quality of resuscitation are realized through the use of contemporary intensive medical techniques, including goal-directed resuscitation strategies, and modern fluid regimens [1]. Even so, a small group of patients may still experience an excessive inflammatory cascade that may result in excessive tissue inflammation and edema, resulting in raised intra-abdominal pressures and potential IAH or an ACS (as much as doubling the patient's mortality). To mitigate this risk, early recognition of the developing ACS remains important (current recommendations continue to suggest routine monitoring of any patient at risk [21]), and prompt intervention, with potential surgical decompression, remains essential.

A novel extension of the damage control surgical strategy in the setting of abdominal sepsis has been the potential avoidance of stoma formation. The hypothesis arose early that, following the physiological restoration of the patient, a patient who otherwise may have received a stoma could avoid this, as a primary anastomosis might now be safely performed in this new physiological milieu. A retrospective, nonrandomized series, and a more recent prospective series, lend support that this approach may indeed be warranted [25, 26].

22.2.3 Intra-abdominal Hemorrhage

Intra-abdominal catastrophes may occasionally result in pure hemorrhagic shock even in the non-traumatic setting. Examples include vascular pathologies such as ruptured aneurysms, ulcer disease, and spontaneous hemorrhage of hepatic tumors or a rupture of a pathological spleen. Similar with other situations, the open abdomen may facilitate a temporizing treatment strategy, or a multistage intervention, where repeated access to the peritoneal cavity is required. It may also avoid or treat an acute abdominal compartment syndrome.

The situation with a ruptured aortic abdominal aneurysm is notable given historical changes. Classically repaired open, patients undergoing the aneurysm repair who developed IAH or an ACS could have a laparotomy fashioned with relative ease. However, more recently, in an surgical era where many of these cases are treated by endoluminal techniques, IAH and ACS may still develop, and a laparotomy may still occasionally be required where nonoperative treatment measures have failed [27, 28]. While the avoidance of a laparotomy to repair the ruptured aorta has assisted the reduction in ACS in this situation, the underlying pathology and hemorrhage may still occasionally manifest with an ACS.

22.2.4 Intra-abdominal Hypertension/Abdominal Compartment Syndrome: Other Causes

The pathological reasons resulting in IAH and ACS are varied and diverse [21]; the underlying causes of the shocked state are fundamentally different. However, the physiological manifestation with increased compartment pressures and eventual tissue and organ compromise is the common outcome. The severe associated morbidity and mortality needs to be avoided by early recognition and institution of appropriate management strategies. The steps have been well described in the published guidelines from the World Society of the Abdominal Compartment Syndrome [21].

The laparotomy offers definitive surgical treatment of the syndrome, particularly where nonoperative measures have failed or have been insufficient. However, as evident from the discussions above, both in the trauma setting and in the situation of severe sepsis, the surgeon may elect to leave the abdomen open prophylactically and potentially prevent the syndrome occurring in the first place. The appropriateness of the risks of an open abdomen compared with the potential benefits needs to be judged in the individual case.

Considerable published clinical experiences exist for select pathologies. For example, in the management of severe acute pancreatitis, the occasional need for an open abdomen is accepted and widely reported [3, 29, 30]. Sixty to eighty percent of patients with severe acute pancreatitis experience IAH, and up to one-third of these develop ACS. Unfortunately, the systemic manifestations of the ACS may be difficult to distinguish from the severe systemic inflammatory response that may occur in these patients. The precise indications and triggers prompting an operative decompression and open abdomen are generally less well documented, as is the influence of patient and environmental factors in this decision. Furthermore, the clinical situation is also changing: of particular interest is an apparent recent reduction in the need for this decompression, perhaps due to more judicious fluid resuscitation regimens, and more intensive monitoring to avoid IAH or an acute ACS.

22.3 Complications of the Open Abdomen

The open abdomen is associated with the development of several unique complications [1–6, 20, 21]. In particular, issues with fluid and protein loss, issues with the loss of the abdominal domain and often associated failure of primary abdominal closure, and issues with fistula formation complicate the management of these patients. These complications impart significant morbidity and mortality to these patients and must be offset by the previously discussed benefits from the laparotomy.

22.3.1 Fluid and Protein Loss

As the peritoneal cavity is left open and drained, ongoing fluid and protein losses will occur. These insensible losses are potentiated by the pathological processes and need to be considered in the overall fluid balance of the patient. As an exudate, the protein losses also must be accounted for; around 2 gm will be lost with every liter of fluid [31].

22.3.2 Loss of Domain/Failure of Primary Closure

Left unchecked, a protracted open abdomen will result in lateral retraction of the abdominal wall and a loss of domain. After around 1 week, the magnitude of this retraction begins to preclude primary abdominal closure and is associated with a rapidly increasing rate of complications during the patient's recovery [1–4]. In a series of laparotomies for traumatic indications, patients closed within 8 days experienced a 12% complication rate, compared with a rate of 52% after 8 days [32]. A recent meta-analysis highlights the worsened mortality, complication rates, and length of hospital stay, among patients that underwent procedures associated with delayed fascial closure; a relative risk of 0.53, $p < 0.001$, with a 12 versus 24% mortality rate, is reported [33]. The importance of an aggressive, early closure plan cannot be emphasized enough.

Every effort to obtain an early fascial closure should be made [1–4]. Ideally this will facilitate a primary closure of the linea alba/rectus sheath. The coordination and planning of the surgical efforts to facilitate this closure need to begin at the time of the index operation where the abdomen is first left open. The type of temporary closure, the nature of the ongoing resuscitation, and the timing of subsequent surgeries are all defining factors. A coordinated strategy should be established by the treating team, optimizing these factors.

A large variety of temporary abdominal closure prostheses/dressings have been described, with many variations developed locally by individual institutions. The historic constructs, such as the plastic silos (e.g., the Bogota bag), while cheap and easily made, are associated with more difficulties regarding wound management and are now clearly shown to be associated with poorer primary fascial closure rates [1–4]. The current standard of care employs a negative pressure wound therapy (NPWT) construct (e.g., with proprietary devices such as the ABThera® system, KCI Acelity, San Antonio, USA) [34]. Apart from the superior wound management and nursing ease, these systems appear to minimize fascial retraction, yielding improved primary fascial closure rates. The NPWT also appears to reduce levels of endotoxin and inflammatory molecules by optimizing the egress of peritoneal fluid containing these pro-inflammatory mediators [35]. In particular, IL-6, IL-10, TNF-α, TNF-β, and CRP all appear in reduced intraperitoneal concentrations. Further research is required, but it is hypothesized that this alteration in the inflammatory milieu may assist the improved outcomes observed in these patients.

Dynamic retention sutures used in conjunction with NPWT appear to modestly further improve primary fascial closure rates. In recent trials, this combination has been reported to have a primary closure rate around 90% [36, 37]. A further study suggested a higher than expected incisional hernia rate (26%), though this issue requires further clarification in studies targeted to address this outcome measure [38].

In patients where a primary closure of the open abdomen is achieved, around 10% may expect an incisional hernia at 21 months [39], appearing similar to other laparotomy hernia rates. This situation will need to be dealt with on its own merits; an optimal treatment strategy will depend on the underlying reasons: infection, fascial necrosis, loss of domain, etc. In any case, a delayed repair, often reinforced with mesh, will be required.

The quality of the resuscitation, both during the initial hospitalization and ongoing during these patients' intensive care treatment, continues to improve [1–4]. Goal-directed therapies, more restrictive fluid prescription algorithms particularly with minimization of crystalloid use, early hemostatic transfusion practices, etc., have all assisted this endeavor. Furthermore, more careful attention to nutrition also appears to be providing results: enteral nutrition appears safe in patients with open abdomens and has been associated with decreased infective complications as well as improved fascial closure rates [1, 31].

Despite best efforts, primary fascial closure will fail to be achieved in some patients. In these cases, the visceral contents will require protection by alternative means: native tissue flaps and grafts, synthetic meshes, and biosynthetic constructs all offer options. Typically, the viscera form an inflammatory cocoon from adhesions, and the laparotomy is dressed and nursed until a temporary mesh closure can be achieved. A delayed definitive abdominal closure is then planned months to years later. As mentioned, these situations are clinically challenging, presenting the treating team with frequent complications to overcome, including fistula formation. A committed multidisciplinary clinical team is required to deal with the complex needs demanded by these patients' pathology.

22.3.3 Fistula Formation

Fistula formation is a well-recognized complication of the open abdomen. It may affect up to one-fifth of patients with an open abdomen, though the risk will vary greatly between patients depending on individual circumstance and the pathology involved [1, 32, 40]. A lack of abdominal wall closure within 8 days, non-protection of the bowel during NPWT, the presence of bowel injury, and repairs or anastomosis are all major risk factors. The degree of sepsis, the number of re-explorations and manipulation of tissues, the quality of the resuscitation, and the use of polypropylene mesh in direct contact with the bowel are also linked [1, 32, 40].

The management of this complication is complex, involving nutritional support, meticulous wound management, and challenging reconstructive and restorative surgeries, and is beyond the scope of this chapter [1, 32, 40]. Naturally, every effort

should be undertaken to avoid such fistulae in the first place; the majority of this will surround optimal patient selection, prosthesis/wound management, and early closure of the open abdomen wherever possible.

22.4 Surgical Judgment

Evident throughout the discussions in this chapter is the crucial role of clinical judgment in the determination of clinical outcomes: patient selection is key. For the optimal management of the open abdomen, ideal patient selection is first and foremost influenced by the patient's physiology [1–6]. However, the subtleties of the pathology and overall treatment strategy, as well as more general patient factors (comorbidities, patient habitus, psychological well-being, etc.), and situational and environmental realities (equipment and resources, local expertise, etc.) are all critical. In the absence of robust, randomized controlled data, this remains a situation for clinical gestalt.

The clinical challenge for the surgeon considering the use of an open abdomen remains the ability to balance this complex, multifactorial equation—an art of weighing the benefits against the potential harm. In an analogous problem to potential overuse of the open abdomen, several authors have recently discussed issues relating to the overuse of the damage control surgical strategy [20, 41]. Simply put, compared with primary definitive operations, damage control laparotomies are associated with increased mortality and morbidity (anastomotic leak, ileus, abdominal wall dehiscence and skin infection, etc.). Therefore, a clinically riskier situation is required to offset these increased costs. This only occurs in select cases that are not amendable or ideally suitable to the primary definitive surgical strategies.

22.5 Future Directions

Significant challenges continue to exist both in the field of basic science research and the clinical realm where these data may translate into clinical care. The quest to understand inflammation remains ongoing; an improved pathophysiological understanding of the inflammatory processes in the shocked patient is urgently needed. An improved understanding of how the different clinical types of shock manifest and alter the patient's normal processes is critical. It may be a worthwhile attempt to broadly classify these critically ill patients into separate physiological groups (trauma, septic, hemorrhagic, etc.). Observations generated from these studies can lead to hypothesis generation which would not only help in identifying the areas for future research and direction but can also help in recognizing those subtle differences between the groups with regard to their physiology, responses, and hence their managements. Within this complex quest, a better understanding of how a patient's individual genetic makeup influences these events also warrants exploration—perhaps genotype-based immune-modulating therapy will one day be an option.

The coordination of future research and scientific efforts may require the assistance of collaborative associations, such as with the World Society of the Abdominal Compartment Syndrome [21]. Such targeted societies achieve to unite clinicians treating otherwise rarely encountered entities, for mutual discussion, and focus each other's research ideas and efforts in joint fashion.

Reflecting on clinical needs in the quest of good care for the open abdomen, the timely and accurate diagnoses of IAH and ACS remain vital. Furthermore, improved methods for screening (i.e., predicting at risk patients) and for the detection of IAH and ACS are still needed. The methods of measuring abdominal pressures, though well described [42], remain clumsy. Apart from the indication of an ACS, similar questions apply in the clinical realm of damage control surgery. Which patients benefit from this surgical mode? If the diagnosis of an ACS is established, or the indication for damage control surgery absolute, substantial clinical questions regarding appropriate intervention (i.e., what approach first, when to use which treatment, how aggressive to intervene, etc.) remain. Regarding the laparotomy, what precisely is the role of a prophylactic open abdomen? Can the risk of developing a compartment syndrome be better estimated? All these clinical questions attempt to master a better understanding of the decision points that influence the tailoring of an optimal surgical strategy. Until these data exist, tailored surgical strategies will remain based on clinical gestalt assisted by the carefully collected current scientific data, combined with our best clinical experience and acumen.

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